ANALYSIS OF NEURO OPHTHALMIC FEATURES OF HEAD TRAUMA

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CERTIFICATE

This is to certify that this dissertation entitled "**Analysis of Neuro Ophthalmic Features of Head Trauma**" has been done by **DR.J.A.Durga** under my guidance in the department of ophthalmology, Madurai Medical College, Madurai.

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DECLARATION

I, Dr.J.A.Durga solemnly declare that the dissertation titled **"Analysis of Neuro Ophthalmic Features of Head Trauma"** has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of master of ophthalmology, Branch III degree Examination to be held in March 2010.

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Date :

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INTRODUCTION

The Darwin's theory of "survival of the fittest" has made man, endeavour to attain higher and still higher speeds in travel and to search for more and still more effective techniques of destruction in war. These have all combined to heighten the incidence of head injuries to an extent unknown to the previous generation.

India has only 1% of the world's automobiles but 6% of road accidents. The death rate per 1000 vehicles is 2.5, which is the highest in the world, even without the recently burgeoning vehicular traffic.

Falls, assault and domestic accidents also account for a significant proportion of head injuries. They affect the active and productive age group in the prime of life.

We often wonder how nature is so resourceful to confine the entire visual system within the brain, but to retain the globe alone to the exterior. This play gifted by nature has, made any insult to the brain in the form of trauma, tumour etc., to be reflected outside by the globe like a mirror. Thus, neuro-opthalmologists and neurosurgeons play essential roles to pick up these neuroopthalmics signs in head injuries, not only to localise the lesion, but also to save the life of the patient and to predict the prognosis.

ANATOMICAL CONSIDERATIONS

Anatomy of visual pathway, ocular motor system and brain relevant to head injury are discussed below.

THE AFFERENT SYSTEM

Anatomy of the optic nerve

The optic nerve is a fibre tract of the brain consisting of a compact bundle of ganglion cell axons, oligodentrocytes and glial cells ensheathed in three meningeal layers. The cranial subarachoid space and the perineural space directly communicate. The subdural space is a potential space which may be occupied by haematoma. The dura which lines the optic canal as periosteum merges with the periosteum of the sphenoidal bone intracranially and splits into two layers, one becoming orbital periosteum and the other remaining as the outer sheath of the nerve.

The average length of the optic nerve is 47-50mm.

It is divided into four parts.

1. **Intraocular part**

The average length of the intra ocular part is 0.7-1mm

It has 4 zones,

1. pars retinalis,
2. Pars choroidalis,
3. Pars scleralis (lamina cribrosa)
4. retrolaminar portion.

The prelaminar part is non-myelinated normally.

2. Intra orbital part

It is 30-35 mm long . It extends from the back of the eye ball to the optic foramen

At the optic foramen it is surrounded by the extraocular muscles originating at the annulus of Zinn. The superior and medial recti origins are adherent to it. The upper and lower divisions of oculomotor nerve, nasociliary nerve, abducent nerve, ophthalmic vein, and ciliary ganglio lie between it and the lateral rectus. Therefore trauma to this region can damage the optic nerve as well as more than one ocular motor nerve and sensory nerves producing the orbital apex syndrome. Orbital fat, the long and short ciliary nerves and arteries surround it. The central retinal artery which enters the nerve 12mm behind its

insertion into the globe is the point of demarcation for anterior and posterior indirect optic nerve injury.

3. **Intracanalicular part**

It is 6-10 mm long .

It lies between the struts at the base of the lesser sphenoidal wing. The optic canal is an average of 9.22mm in length and contains the optic nerve, meninges, ophthalmic artery and the post ganglionic sympathetic fibres. The Dura of the nerve sheath is fixed to the periosteum and bone of the canal.

The medial wall of the canal is on average 0.21 mm thick and is absent in 4% of normal persona, the dural sheath being in contact with the sphenoidal sinus mucosa.

The distal portion of the canal, closest to the orbit has the thickest wall and narrowest cross section.

4. Intracranial part (10mm):

This part of the optic nerve is about 10mm in length and lies above the cavernous sinus and converges with its fellow (over the diaphagma sellae) to form the optic chiasma.

Relations

Superior	:	Anterior perforated substance, olfactory tract, Anterior cerebral artery
Inferior	:	Diaphragma sellae, cavernous sinus

Lateral	:	Internal carotid artery
Medial	:	Ophthalmic artery

Optic Chiasma

The two optic nerves end in optic chiasma. The nasal fibres decussate. The temporal fibres do not decussate.

Optic Tract

The optic chiasma continues as optic tracts each optic tract carrying the temporal fibres of the same eye and the nasal fibres of the opposite eye.

Lateral Geniculate Body (LGB)

The optic tracts end in LGB from where the III order neuron starts.

The pupillary fibres from the optic tract go to the pretectal nuclei without going to the LGB.

The optic radiations from the LGB terminate in the visual cortex in the occipital lobe (Striate cortex) (area 17) and visual association areas are (Area 18 and Area 19) parastriate and peristrate cortex respectively.

THE EFFERENT SYSTEM

Neural basis of eye movements

Types of eye movement

Fast Eye Movements

(velocity:300°-700°/sec)

1. Saccades Spontaneous

2. Nystagmus quick phase

3. REM in sleep (Rapid eye movement)

Slow Eye Movements

(velocity:20°-50°/sec)

1. Voluntary smooth pursuit
2. Vergence
3. Optokinetic reflex
4. Vestibulo-optic reflex

Premotor Command

Horizontal Movements : Paramedian Pontine Reticular formation

Vertical Movements : Rostral mesencephalic reticular formation

[incl. Rostral insterstitial nucleus of medial longitudinal fasciculus and Intermediate nucleus of cajal].

These nuclei project to specific ocular motor nuclear subtroups and receive afferents from the cortex, vestibular system, cerebellum, superior colliculi etc.

Cortical areas

1. Area striata of occipital cortex (area 17) has various visuomotor connections, concerned with fixation reflexes.
2. Frontal eye field
    - It is situated at the posterior middle frontal gyrus, area 8
    - It is concerned with contralateral conjugate gaze
    - Damage may cause ipsilateral gaze deviation (or) poor contralateral gaze but normal reflex movements.
3. Parietal cortex [area 7]

    - It is related to attention to visual targets and smooth pursuit. Bipareital lesions produce ocular motor apraxia.

Cerebellum

It is the major coordinator for movements including eye movements. It is receives afferents from visual and vestibular apparatus, neck and proprioceptors. It projects to supranuclear zones concerned with ocular movements, both smooth pursuit and saccades.

Disorders cause nystagmus, ocular dysmetria, opsoclonus or ocular flutter.

Medial longitudinal bundle

This white matter tract extends from the rostral midbrain to the spinal cord. It is in close connection to ocular motor and other cranial nerve nuclei.

It mediates correlation between all three ocular motor nuclei and all four vestibular nuclei. Lesions produce internuclear ophthalmoplegia.

OCULAR MOTOR NERVES

Oculomotor nerve (The III cranial nerve):

Nucleus: It is situated in the midbrain at the level of superior colliculus.

Courses: It passes forwards between posterior cerebral and superior cerebellar arteries and laterally parallel to posterior communicating artery. On lateral side of posterior clinoid process, it pierces the dura and enters lateral wall of cavernous sinus, above the IV cranial nerve. It divides into superior and inferior divisions which enter the orbit through superior orbital fissure within the tendinous ring.

Muscles supplied: Superior rectus, inferior rectus, medial rectus, inferior oblique and levator palpebrae superioris.

Trochlear Nerve (The IV cranial nerve)

Peculiarities

This is the only cranial nerve to arise from the dorsal aspect of the brain.

This is the longest and thinnest of all cranial nerves

Nucleus: It is situated in the midbrain at the level of inferior colliculus.

Course: It emerges on posterior surface of brain stem, decussates and passes forward around the cerebral peduncle. It enters the lateral wall of the cavernous sinus and lies below the III Cranial Nerve and above the first

division of V Cranial nerve. It passes through the upper part of superior orbital fissure, outside the tendinous ring.

Muscle supplied : Superior oblique muscle.

Abducens Nerve (The VI Cranial Nerve)

Nucleus: It lies in upper part of the floor of the fourth ventricle.

Course: It emerges between the pons and medulla oblongata, runs upward, forward and laterally, makes an acute bend across the sharp border of petrous part of the temporal bone and run within the cavernous sinus inferolateral to the Internal carotid artery. It enters the orbit through the superior orbital fissure between the two divisions of the III Cranial nerve.

Muscle supplied: Lateral rectus muscle.

THE PATHOPHYSIOLOGY OF HEAD TRAUMA

Occurrence of ophthalmological complications of head injury depends on

1. Brain trauma itself [concussion contusion, laceration] with visual complications
2. Skull fracture in the occiput or base with damage to the visual cortex or the nerves subserving the eyes.
3. Cerebral compression due to meningeal haemorrhage [acute or chronic, extradural, subdural, subarachnoid or intracerebral]

Progressive cerebral compression due to intracranial bleed goes through four stages:

Harvery Cushing proposed the following stages:

1. Stage of physical compensation: CSF displaced, BP rises, and circulation maintained–symptomless.
2. Stage of venous compression – irritative signs appear.
3. Stage of capillary Anaemia – paralytic signs appear.
4. Irreversible stage of perivascular haemorrhages and progressive brain edema.

Consequences of a space occupying lesion (SOL)

A) Herniation of the cingulated gyrus under the falx

B) Tentorial herniation of the parahippocampal gyrus

C) Herniation of cerebellar tonsil through the foramen magnum

D) Impingement of the crus against the tentorium

E) Haemorrhage into the midbrain

BIOMECHANICS OF BRAIN INJURY

By definition head trauma is an anatomical damage or physiological disturbance caused by application of mechanical force to the head.

Therefore the nature, site and magnitude of the mechanical force are the main determinants of head injury. The modification or elimination of this force is the major preventive strategy.

Primary brain injury

1. **Contact Injuries**
2. **Inertial Injuries**

Contact injuries

A direct impact causes complex mechanical events both near and distant to the point of contact. They typically cause focal injuries and do not cause diffuse brain injury. Contact forces produce injury in the immediate vicinity of skull injury. Since most impacts set the skull in motion, there are associated acceleration injuries also.

Local contact Effects

1. Depressed fractures
2. Linear fractures
3. Basilar skull fractures
4. Extradural haematoma
5. Coup contusions

Remote contact effects

Remote contact effects are caused by skull deformation and stress waves transmitted at high velocity

1. Remote linear fractures (vault or base)
2. Remote brain contusions and haematoma
3. Brain herniations caused by transient distortion, classically in infant skulls.

Small high velocity objects may shutter the skull and drive bone fragments deeply.

When the surface of contact is larger or the skull hits a fixed surface the result depends on the nature of mechanical loading.

Types of Mechanical Loading

	Static loading	Dynamic Loading
Incidence	Less common	Common
Force duration	Input force applied in a period >200ms	Input force applied in less than 50ms
Examples	Slow moving vehicles	Most RTAs, falls from height

Dynamic Loading

In dynamic loading there may be contact forces or inertial forces. Accordingly there are two subtypes of dynamic loading.

1. Impulsive loading
2. Impact loading

Impulsive loading

- Only inertial forces
- The head is accelerated or decelerated without directly being struck
- For example, blow to face or thorax, or fall from height

Impact loading

- Contact forces + inertial forces
- A short impact to head accelerates or decelerates it.
- Local skull injury in common

Inertial injuries

Head acceleration causes structural or functional damage to neural and vascular structure by the relative movement of the brain in relation to the dura and skull. They cause concussion, contusion and diffuse axonal injuries in the brain tissue.

The types of acceleration are

1. Translational
2. Rotational
3. Angular i.e. translational and rotational

Angular acceleration is most commonly encountered. The center of angulation is usually the midcervical spine.

Angular acceleration is the most injurious as it causes shear strain deformation because of differential motion of one portion of the brain with respect to another.

Secondary cerebral injury

In contrast to primary brain injury which occurs at the movement of impact, secondary cerebral injury is the result of intracranial space occupying lesion, either by oedema or haematoma.

Many of the effects of secondary damage are preventable by active management of head injuries in the early stages and by a close observation of clinical signs.

Type of primary lesion	**Degree of tissue damage**	**Resulting space occupying lesion**
1) Diffuse axonal injury & contusion	+++	Oedema
2)Subdural	+	Haematoma
3) Extradural	+	Haematoma
4) Intracerebral	++	Haeamatoma

Important Note:

Senile cerebral atrophy favours increased shear deformation and also subdudal haemorrhage.

Correlation of brain lesions and injury mechanism

Brain Lesion	Mechanism of injury
1) Skull fractures, epidural haematoma coup contusion	Contact forces
2) Contrecoup contusions	Inertial effects (impact or impulsive)
3) Intra cerebral haematoma	Stress waves and acceleration induced strains
4) Subdural Haematoma	Bridging vein disruption
5) Cerebral concussion	Angular accelerations with electrophysiologic dysfunction
6) Diffuse axonal injury	Long duration angular acceleration esp. in coronal plane

THE NEURO OPHTHALMIC SIGNS IN HEAD TRAUMA

Neuro ophthalmic signs following head trauma

It is divided into those that occur during

1. Acute phase
2. Chronic phase

Acute phase of head injury involves

1. Eye position
2. Eye Movements
3. Pupillary signs
4. Cranial nerve injuries
5. Cortical blindness

Chronic phase of head injury involves

1. Delayed III & VI nerve palsies
2. Post-traumatic papilledema / Optic atrophy
3. Post-traumatic field defects
4. Aberrant regeneration of the Cranial nerves
5. Vascular complications
6. Infections

EYE POSITION IN UNCONSCIOUS PATIENTS

Abnormal position of the eyes in an unconscious patient is indicative of structural damage to the brain and the brainstem.

- Unilaterally abducted eye indicates unilateral tentorial herniation.
- Bilaterally abducted eyes are suggestive of central herniation.
- Skew deviation of the eyes is produced by posterior fossa lesion.
- Persistent gaze deviation is caused by
    - Ipsilateral frontal eye field damage or
    - Contralateral pontine gaze centre damage.
- Persistent down gaze deviation with upward gaze palsy occurs when there is posterior transtentorial herniation with tectal compression. It is called Parinaud syndrome / dorsal midbrain syndrome.

EYE MOVEMENTS

Intact or absent eye movements in unconscious head injured patients reflect the integrity of the brainstem function. Absence of eye movements indicates that there is primary or secondary (due to herniations) injury to the brainstem and therefore is a poor prognostic sign.

Injury to the cervical spines should be ruled out before eliciting the following reflex eye movements.

Oculocephalic reflex: (“Doll’s Eye Movements”)

In an unconscious patient, when the head is turned to one side the eyes will deviate to the opposite side. There will not be any movements if the brainstem is damaged.

Oculo-vestibular reflex

This test is performed by syringing the ears (with intact drums) with cold water (30°C) or warm water (44°C). When cold water is used, the eyes will deviate to the same side and nystagmus occurs in the opposite direction. With warm water, the eyes will deviate to the opposite side and the direction of the nystagmus would be to the side of syringing.(cold - opposite, warm – same COWS)

PUPILLARY SIGNS FOLLOWING

HEAD TRAUMA

Examination of the pupils is a simple and the most reliable test in head trauma. Involvement of pupils rules out a metabolic cause of coma.

I. Bilaterally Dilated pupils

In a semiconscious irritable patient, dilated but reacting pupils may be caused by anoxia. This is usually temporary. The pupils become normal on oxygen administration.

In a deeply comatosed patient, bilateral dilated and fixed pupils indicate severe injury to the brainstem.

Primary injury to the midbrain may produce mid dilated sluggishly reacting pupils due to lesions of both sympathetic and parasympathetic tracts.

II. Unilaterally dilated pupil

This may occur due to

1. Transtentorial herniation [coning]
2. 3rd nerve palsy
3. Traumatic mydriasis

1. A widely dilated, immobile, Hutchinsonian pupil / cook's pupil

There are 3 stages in its evolution.

1. An initial miosis from ipsilateral nerve irritation
2. A dilatation of ipsilateral pupil which still reacts to light and convergence while the patient may be drowsy.
3. A true Hutchinsonian pupil, a unilateral dilated fixed pupil, patient shows increasing drowsiness to produce coma.

Mechanism

A space occupying hematoma / expanding brain edema may lead to herniation of temporal lobe into the tentorial hiatus to impinge directly on the III nerve. It is associated with contralateral motor signs. It needs urgent decompression to prevent damage to the brainstem which may have grave prognosis.

Kernohan notch syndrome

Occasionally pupillary and motor signs are ipsilateral due to cross compression of opposite cerebral peduncle.

2. III Nerve palsy

Patient has dilation of pupil, ptosis, paralysis of adduction, elevation and depression. Patient is usually conscious.

3. Traumatic mydriasis

There will be evidence of injury in and around the orbit. This may be due to injury to

1. iris
2. ciliary ganglion
3. short ciliary nerve

III. Bilateral constricted pupils

Intrinsic pontine lesions produced by contusion or haematoma cause bilateral small constricted pupils [pin-point pupils or “pontine” pupils’. This is due to the injury to bilateral sympathetic nerves. Pupillary reaction to light can be observed if magnifying lens is used.

IV. Unilateral constricted pupils

It is caused by oculosympathetic paralysis that is Horner’s syndrome. Other sign are, partial ptosis enophthalmos and anhidrosis.

Marcus-Gunn pupil [Relative afferent pupillary defect]

It is seen in optic neuropathy. It is ellicited by swinging flash light test. On throwing the torch light to normal eye both the pupils will constrict. Upon swinging the light to the abnormal eye, both the pupils will paradoxically dilate.

TRAUMATIC OPTIC NEUROPATHY [TON]

Traumatic optic neuropathy is divided into 3 categories

1. Avulsion
2. Direct injury
3. Indirect injury

Avulsion

Avulsion is the rarest form of optic neuropathy. Total avulsion implies complete separation of the lamina cribrosa from its attachment to the sclera. The choroids, retina and vitreous are completely separated from the optic disc. The retinal blood vessels are partly or totally interrupted.

Partial avulsion involves a localized segment of the optic nerve. Optic nerve avulsion may be caused by penetrating or non-penetrating injuries. Severe orbital fracture due to blunt trauma is the commonest cause.

3 mechanisms postulated

1. Increased intraocular pressure due to the globe being compressed against the bony orbit and the optic nerve being pushed out of the scleral canal.
2. Increased intraorbital pressure forcing the globe forward, stretching and tearing the optic nerve.
3. Extreme rotation and displacement of the globe within the orbit.

Visual progrnosis depends on the extent of avulsion and total blindness occurs in complete avulsion.

Immediately after injury the optic disc is obscured by an overlying vitreous hemorrhage. Associated ocular findings are subconjunctival haemorrhage, limitation of extraocular movements, proptosis and a dilated, fixed pupil.

When the fundus view improves, the disc contour is seen distorted or the scleral canal devoid of the disc tissue is seen. Subsequently the defect is closed by a gliotic scar which may extend into the vitreous.

Direct Optic Nerve Injury Mechanisms

Lacerations

- Incomplete
- Complete

Bone fracture or deformation

- Optic canal
- Orbital walls
- Anterior clinoid process

Vascular Insufficiency

- Ischaemia
- Infarction

Haemorrhage

- Nerve sheath
- Intra neural

Direct optic nerve injury is due to impingement of the nerve by either

(1) A foreign body that has penetrated the globe, orbit or cranium or

(2) Injury from a displaced fracture or spicule of bone in the region of the optic canal.

Types of direct injury

Anterior type

If the damage is anterior to the entrance of the central retinal artery the ophthalmoscopic picture is of central retinal artery occlusion, and lose of vision is instantaneous and complete.

Posterior type

More posterior injury may demonstrate a normal fundus or mid retinal oedema due to axoplasmic statsis. Features of primary optic atrophy appear in 3 to 4 weeks.

Indirect optic nerve injury

It is the commonest form of traumatic optic neuropathy.

Indirect trauma most commonly affects the intracancalicular portion of the optic nerve.

Pathophysiology

Three mechanisms are postulated

Vascular insufficiency is the common factor:

1. Shear causes rupture, spasm or thrombosis of small arterioles which leads to haemorrhage, infarction and oedema compressing the nerve in the canal causing further ischaemia.
2. Shear causes oedema, raised intracanalicular pressure, compression and ischemia.
3. Nerve haematoma: Subdural / intradural / subarachnoid

Holographic interferometry shows stress, for surface loads on frontal and malar eminence are transmitted to optic foremen even without fracture.

In closed head trauma, the intracanalicular portion of the optic nerve, being fixed is commonly damaged.

The intraocular segment is occasionally damaged.

The intraorbital and intracranial portions due to relative mobility and laxity are usually spared.

Types of indirect injury

1. Anterior type - Fundus abnormalities are present
2. Posterior type - Fundus normal is initially

Anterior type

The intraorbital segment containing the central retinal artery is affected and the fundus picture resembles central retinal artery occlusion. Disc edema with normal retinal circulation and fluorescein angiographic findings of impaired posterior ciliary artery circulation is another form.

Lastly small anterior marginal tears of the disc with small haemorrhages less than one-third disc circumference, which resolves in two weeks with a heavily pigemented scar, followed 1 month later by mild disc pallor may occur.

Posterior type

When the optic nerve dysfunction is associated with a normal fundus initially and no evidence of chiasmal lesion then the lesion is presumed to lie in between the central retinal artery entrance and the chiasm. Disc pallor retinal nerve fibre layer defects become apparent 4 to 8 after head injury.

Clinical investigations in indirect injury

1. Every effort should be taken to evaluate visual function
2. Snellen, rosenbaum near card should be used
3. If the patient does not have perception of light, projection should be checked in all the four quadrants.
4. Pupillary responses are critical. Grading of RAPD should be done with neutral density filters.

Pupillary response to light is the most reliable sign of the extent of optic nerve injury.

5. Field: Central scotoma or NFL defects
6. Direct / Indirect ophthalmoscopy: Initially it may be normal
7. VER – Adjunctive Test. There is the good correlation between initial VER and ultimate acuity especially in comatose patients.

Chiasmal lesions

The chiasm bears a close relationship to the III ventricle and hypothalamus. It is damaged relatively rarely in severe frontal trauma due to acute traumatic necrosis, ruptured pial vessels or direct damage to nerve fibres. This causes bitemporal hemianopia with hypothalamic symptoms.

Optic tract lesions

Usually the severity of injury precludes the survival.

- Theoretically a retrochiasmatic, tract lesion causes contralateral homonymous heminopia, which is incongruous if incomplete with Wericke's hemianopic pupil.

Lateral geniculate body lesion

Field defect, as for tract or a relatively congruous sectoranopia. It is extremely unlikely to be damaged in isolation in head injury.

Optic radiation lesion

It causes quadrantanopia to hemianopia – usually congruous

Visual cortex lesion

It causes congruous hemianopia.

Concussion and edema may cause reversible cortical blindness. The posterior occipital pole containing the macular area is susceptible to occipital trauma. It is also the watershed zone between the posterior cerebral artery and middle cerebral artery territories and so susceptible to ischaemia caused by hypovolemic shock and generalized hypoperfusion state. A central homonymous hemianopic scotoma results.

OCULAR MOTOR DISTURBANCES

Ocular motor disturbances following head injury apart from those caused by direct damage to muscles and nerves in the orbit account for 15% of all ocular motor palsies, either occurring due to birth trauma or accidents later in life. They complicate 1% of accidental trauma to the head.

Clinical presentation

A complete spectrum of clinical presentations are reported from partial paresis of individual nerves to bilateral total ophthalmoplegia.

Supranuclear palsy

Supra nuclear ocular motor palsies are rare.

They cause conjugate gaze deviations where there is retention of ocular parallelism and absence of diplopia.

Pontine lesion : It causes horizontal gaze palsy

Midbrain lesion : It causes vertical gaze palsy

Cerebral lesions

Cerebral lesions may cause

1) Conjugate palsies, often temporary with exaggerated fixation reflexes.
2) Sparm of conjugate gaze to the side opposite the lesion.

The psycho-optical reflexes – fixation, convergence, blinking etc. may be disoriented. Nystagmus may occur due to vestibular or cerebellar involvement.

Defects of convergence and accommodation may occur which frequently represent unmasking of a previous phoria and often perpetuated as a neurosis.

CRANIAL NERVE INJURIES

The exact incidence of damage to each cranial nerve varies with patient selection and length of follow up. Olfactory, facial and audito vestibular nerves are damaged most often by blunt head trauma.

III Nerve injury

Trauma is one of the most common causes of third nerve palsy and accounts for 20% of ocular motor injury. Anisocoria due to orbital trauma has to be distinguished.

The lesions may be in the nucleus or root which is associated with brainstem damage or in the trunk due to basal fractures or haematoma compression. Stretching and concussion may cause temporary lesions, and laceration or ischaemia cause permanent damage.

Spontaneous recovery takes 4-6 months. Aberrant regeneration is well documented in the following frequency:

1. On down gaze upper lid retracts – it is called Pseudo Von Graefe Sign
2. Constriction of the pupil associated with lid retraction.
3. Widening of the palperbralfissure on adduction, narrowing on abduction – It is called Pseudo Duane Syndrome
4. Dilated pupil unreactive to light but constricts with convergence / adduction – It is called Pseudo Argyll Robertson Pupil.

IV Nerve injury

Traumatic IV nerve injuries are usually diagnosed when the patient recovers consciousness and experiences diplopia. The usual head posture is tilt to the opposite side, with hypertropia of the affected eye.

Park's Bielschowsky three step test:

This test is useful in the diagnosis of fourth nerve palsy.

Step 1 : In primary position in the affected side is hypertropic

Step 2 : On lateral gaze, hypertropia worsens on opposite gaze. (WOOG)

Step 3: On head tilt, hypertropia becomes better on opposite tilt. (BOOT)

Trauma accounts for 29% percent of all IV nerve injuries.

Bilateral fourth nerve palsy is nearly always post-tramatic.

VI Nerve injuries

Minor degrees of abduction deficit are often ambiguous after head injury. Bilateral VI nerve palsies are relatively common.

Head trauma is the cause of 15% of VI nerve palsy.

Facial nerve injury

The facial nerve may be implicated in cranial trauma due to

1. Fracture of the petrous temporal bone frequently associated with ear bleed, deafness and vestibular disturbance (nausea, tinnitus, nystagmus,

diplopia). A transverse fracture causes immediate palsy: A longitudinal fracture causes delayed palsy.

2. Direct trauma near the stylo-mastoid foramen,
3. Displaced fracture of the mandible, or
4. Penetrating trauma or contusion of the nerve branches.

Epiphora, lagophthalmos and paralytic ectropion result with exposure keratitis if unrecognized.

Localizing signs

Facial nerve injury between the 6^{th} nerve nucleus and the geniculate ganglion involves fibres destined for the greater superficial petrosal nerve and impaires lacrimation, whereas distal lesions do not.

The intermediate nerve of Wrisberg which synapses in the geniculate ganglion and leaves the facial nerve 3 to 4mm above the stylomastoid foramen innervates salivary secretion and taste to the anterior 2/3 of the tongue. Involvement of the stapedius nerve in a fracture may also cause hyperacousis.

Healing may be associated with a number of aberrant regeneration syndromes, the classic one being a paradoxical gustatory lacrimal reflex [Crocodile tears].

Trigeminal nerve involvement

This may occur intracerebrally or at the skull base causing anesthesia over the area subserved (including cornea) by the affected branch. It is usually associated with some ocular motor palsy.

Vascular complications

Terson syndrome

Rarely, subarachnoid hemorrhage will lead to papilledema, retinal hemorrhage and ophthalmoplegia.

Carotid cavernous fistula

It rarely occurs due to direct damage to the vessel wall. It is a late complication of head trauma. It is characterized by abrupt onset of pain, a noise felt in the head by the patient and pulsating proptosis and hypoxic eye ball syndrome.

Chronic phase of head injury

Delayed 6^{th} and 3^{rd} nerve palsies

Delayed VI nerve palsy often results from increased intracranial pressure or haemorrhagic meningitis. Delayed III nerve damage, even without pupillary involvement is a more ominous sign, indicating trastentorial herniation.

Post-compression blindness

A few patients who survive intracranial haematomas and brain oedema, complain of defective vision. This type of visual failure results from two mechanisms.

(i) When there is diffuse oedema of the brain, the medial and inferior portions of the frontal lobes (gyrus rectus) swell and compress the optic nerves and the chiasma, resulting in optic atrophy.

(ii) During transtentorial herniation the medial portion of the temporal lobe compresses the 3rd nerve as well as the posteior cerebral artery. This results in ischaemia of the occipital lobe and a homonymous field defect.

Post-traumatic optic atrophy

The causes are:

1. Traumatic optic neuropathy
2. Injury to the optic chiasm
3. Optico-chiasmal arachnoiditis
4. Post-papilloedemic optic atrophy

Post-traumatic papilloedema

Any post-traumatic lesion which causes a sustained increase in intracranial pressure for 2 to 3 weeks can produce papilloedema. The usual causes are:

1. Chronic extradural haematoma
2. Chronic subdural haematoma
3. Communicating hydrocephalus caused by adhesions following subarachnoid haemorrhage.
4. Post-traumatic brain abscess
5. Incomplete occlusion of major venous sinuses.

Post-traumatic field defects

The optic radiations in the temporal and parietal lobes may get injured by deep contusions or intracerebral haematomas. The occipital poles may themselves get contused in coup and contre-coup injuries. These type of injuries will produce homonymous field defects (when one of the occipital lobes is involved) Incomplete injuries to the optic chiasma cause bitemporal field defects. Post-traumatic optico-chiasmal arachnoiditis produces irregular field defects.

MANAGEMENT OF HEAD TRAUMA

Initial Assessment and resuscitation forces on airway, breathing and circulations. Initial blood loss is replaced by crystalloid till the site of bleeding is located.

Neurological Assessment

Accurate history regarding the mode of injury, loss of consciousness is obtained. Assessment should include a gauge of level of consciousness as well as focal neurological deficit. The Glasgow Coma Scale grades eye opening, best motor response and verbal response on a 15 point scale. Blood pressure, respiratory rate should be recorded. Pupillary response should be noted. It has a high degree of interobserver consistency.

Skull Radiology

77% of adults and 62% children who develop haematoma have skull fractures. CT should be performed if

(1) There is a depressed skull fracture

(2) Focal neurological signs

(3) Deterioration of consciousness or coma

(4) Not regained consciousness despite adequate resuscitation

(5) Post traumatic seizure.

Radiological findings following head injury

Skull fracture if close to vascular markings should alert the observer to the risk of intracranial haematoma. Opacity of air sinsuses or pneumocephalus indicate a compound fracture. CT gives an accurate picture of the nature and extent of any bone injury and it is an accurate method of detecting an intracranial bleed. Fresh blood may be of mixed intensity but haematoma appears hyperdense. Extradural haematoma is convex, subdural haematoma is concave. Subdural haematoma is usually associated with brain contusion, if present alone, a primary vascular cause for injury should be suspected. MRI is superior to CT to detect soft tissue injuries.

Medical management

The aim is to prevent secondary changes and optimize recovery from the primary injury. When a patient needs ventilation the aim is to control ICP by preventing fluctuations in oxygenation and circulating volume, preferably with continuous ICP monitory. Raised ICP is controlled using IV mannitol, frusemide and hyperventilation.

Surgical Management of hematoma

In the absence of CT, when patient is rapidly deteriorating the site of a skull fracture is the first guide for burr hole placement particularly if it is close to a vascular marking, otherwise CT guides the location.

Management of traumatic optic neuropathy

In the initial stage, pulse steroid therapy in the form of IV methyl prednisolne 1g in 100ml of NS infused over 15-20 min given OD for 3 days followed by oral predinisolone 1mg/kg/day for 11 days followed by rapid tapering over the next 3 days.

If there is no response, transethmoidal decompression of the canalicular portion of the optic nerve should be attempted.

Deviation of eye

Diplopia secondary to ocular motor nerve palsies are treated with prisms and muscle surgeries based on the severity and duration of paralysis.

Management of complications

Infections like meningitis/ cerebral abscess should be treated with IV broad spectrum antibiotics.

Carotid Cavernous Fistula → Detachable flow guided balloon, to close the CCF.

REVIEW OF LITERATURE

1. **Sabates N, Gonce M, Farris B. Neuro-ophthalmological findings in closed head trauma. J Clin Neuroophthalmol 1991;11:273-7.**

Sabales et al in 1991 conducted a study with 181 cases and found that loss of consciousness and ocular motor nerve palsy are significantly correlated statistically. Lack of seat belt and neuro ophthalmic abnormally were also correlated significantly.

2. **Jacobi et al in 1986 examined 741 cases of head trauma and inferred that the most common cranial nerve affected was oculomotor nerve.**

Jacobi G, Ritz A, Emrich R. cranial nerve damage after paediatric head trauma: a long-term follow-up study of 741 cases. Acta Paediatr Hung 1986;27:173-87.

3. Gjerris in 1976 quoted that approximately 5% of all patients with head trauma. Manifest injury to some portion of visualpthaway.

4. **Mariak Z, Stankiewicz A. Cranial nerve II-VII injuries in fatal closed head trauma. Eur J Ophthalmol 1997;7:68-72.**

Mariak et al found positive correlation between avulsion of III cranial nerve and basilar skull fracture in 1997.

5. **Keane J. Neurologic eye signs following motor vehicle accidents. Arch Neurol 1989; 46:761-2.**

Keane et al examined 96 patients in his study in 1984. He revealed that the most common mode of injury was motor vehicle accident [97%].

6. **Moster M, Volpe NJ, Kresloff MS. Neuro-opthalmic findings in head injury. Neurology 1999;52 (suppl 2): A23.**

Moster et al in 1998 examined 46 head trauma patients in rehabilitation and found the III nerve as the most common efferent nerve injuried.

7. **Sabates N, Gonce M, Farris B. Neuro-ophthalmological findings in closed head trauma. J Clin Neuroophthalmol 1991;11:273-7.**

Sabales et al found that the mean age of patient with head trauma was 31 years and functional visual field defect in the most common afferent defect.

8. **Lepore F. Disorders of ocular motility following head trauma. Arch Neurol 1995;52:924-6.**

Lepore in 1995 studied 60 head trauma patients and inferred that the most common efferent deficit was IV nerve palsy.

9. **Gregory P. Van Stavern Neuroophthalmic manifestations of head trauma. Journal of Neuro-ophthalmic 21(2):112-117, 2001.**

Van Stavern et al in his study in 2000 concluded that retrochiasmal visual field defect was the commonest afferent visual defect.

10. **Kowal L. Ophthalmic manifestations of head injury. Aust N Z J Ophthalmol 1999;20:35-40.**

Kowal et al in 1992 in his study with 161 patients found optic neuropathy as the most common afferent defect among head injured patients.

11. Gregory P. Van Stavern Neuroophthalmic manifestations of head trauma. Journal of Neuro-ophthalmic 21(2):112-117, 2001.

Van Stavern et al found abnormal neuro imaging studies only in 1/3rd of patients with significant neuroopthalmic deficit.

12. Lepore F. Disorders of ocular motility following head trauma. Arch Neurol 1995;52:924-6.

Lapore et al in 1995 found statistically significant correlation between bilateral cranial nerve palsies and corticospinal tract damage.

AIM OF THE STUDY

1. To determine the mode of injury in head injury patients.
2. To document the incidence and nature of neuroopthalmic deficits in head injury patients.
3. To correlate the initial level of consciousness and the incidence of neuro-opthalmic deficits.
4. To correlate the documented neuro-ophthalmic deficits with the neuroimaging studies.
5. To analyse the recovery pattern of the head injured patients.

MATERIALS AND METHODS

A clinical study was carried out in the Department of Ophthalmology. Government Rajaji Hospital, Madurai, during the period from May 2008 to October 2009.

Out of 1086 patients admitted in head injury ward, 182 patients with the history of head injury were referred from the department of nuerosurgery who had ophthalmic complaints. Of them 100 patients were found to have abnormal neuroopthalmic deficits.

Inclusion criteria

1. Patients with definite history of head trauma.
2. Patients with ophthalmic symptoms.
3. Patients whose level of consciousness is good with or without history of loss of consciousness in the immediate post-head trauma period.

Exclusion Criteria

1. Unconscious patients who did not subsequently recover adequate consciousness.

2. Patients whose neuro-ophthalmic deficits were not confirmed or follow up was not possible due to default [Against Medical Advice/Absconded]

The cases were examined in the Department of Neurosurgery and in the Neuro-ophthalmology Clinic in the Department of Ophthalmology subsequently.

All the data were collected or a standardized proforma and wherever appropriate slit lamp examination, indirect ophthalmoscopy, diplopia charting, forced duction tests visual field analysis etc were done.

In cases requiring ophthalmic medical or surgical intervention, they were instituted appropriate work-up in the ophthalmology Dept., All the cases were essentially managed in the Department of Neurosurgery with routine follow up.

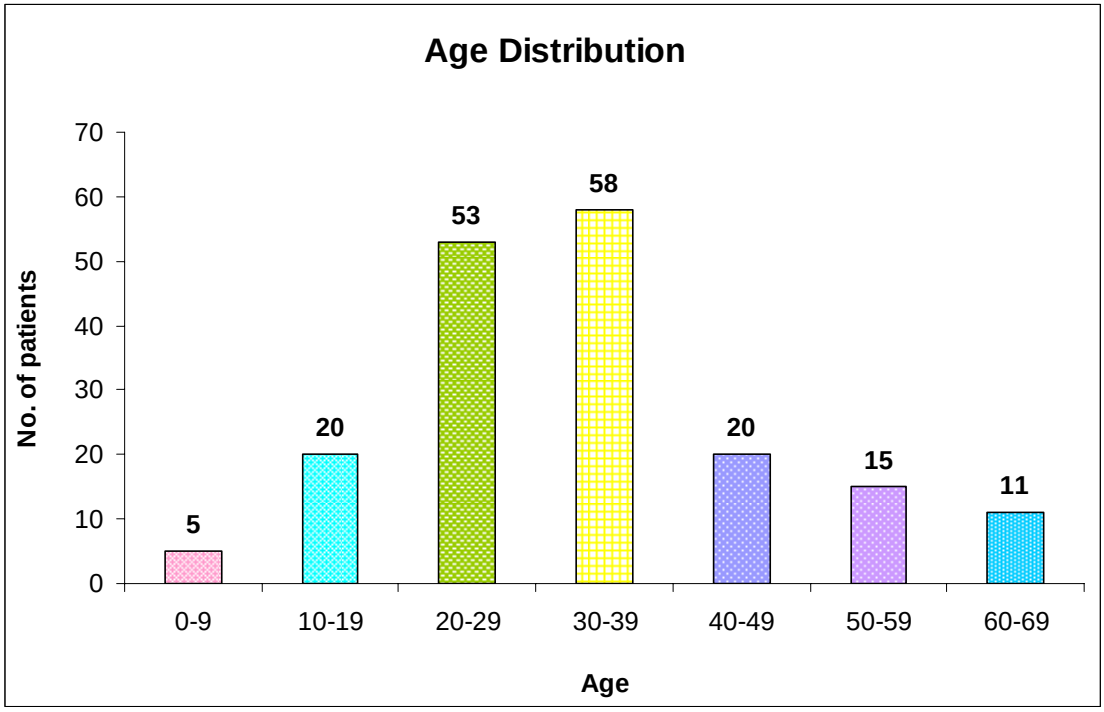
All the patients were followed up for a minimum period of 3 months. They were reexamined and the results were recorded regarding the original problem and any new findings.

OBSERVATIONS AND ANALYSIS

I. Age distribution

A total of 182 patients were examined. The age range was 2-70 years with a median of 32 years. Maximum number of head injury patients were in the age group of 20-40 years. Least number of patients were in the extremes of age.

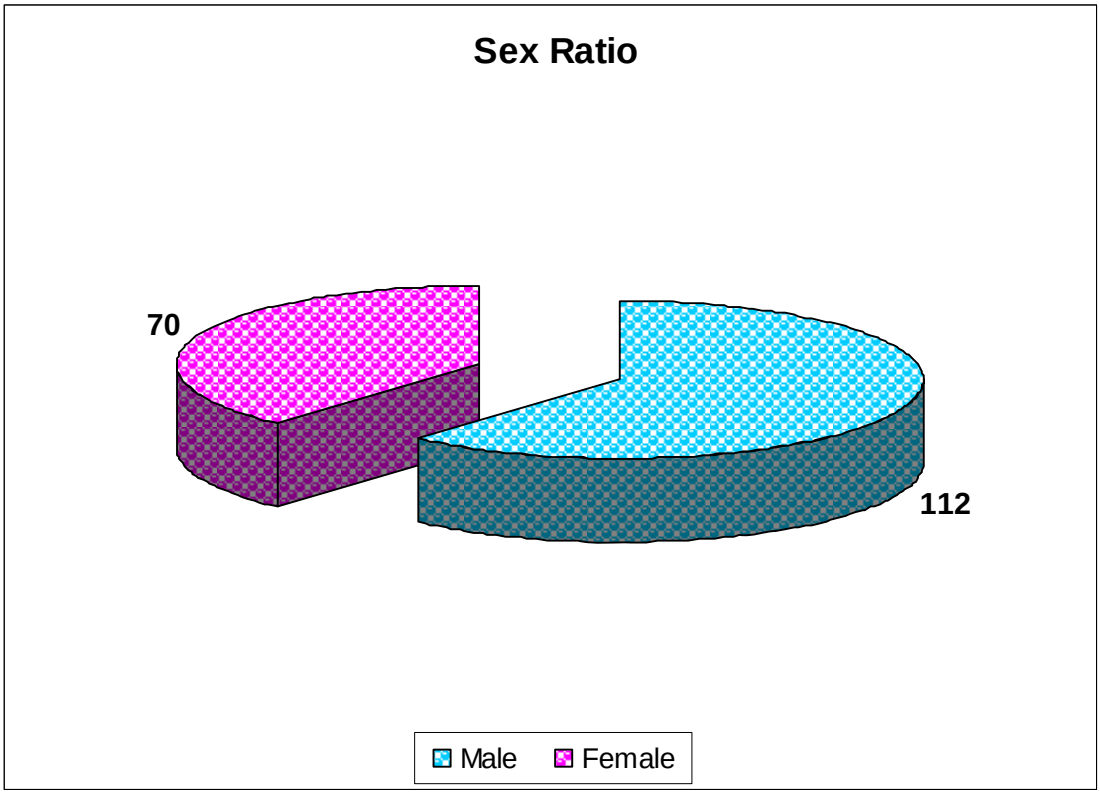
Age range (in years)	Number of patients	Percentage (%)
0-9	5	2.74
10-19	20	10.99
20-29	53	29.12
30-39	58	31.87
40-49	20	10.99
50-59	15	8.24
60-69	11	6.04


Age Distribution
No. of patients
70
60
50
40
30
20
10
0
5
20
53
58
20
15
11
0-9
10-19
20-29
30-39
40-49
50-59
60-69
Age

II. Sex Ratio

Of 182 patients, 112 patients were males and 70 were females. M:F ratio was 1.6:1.

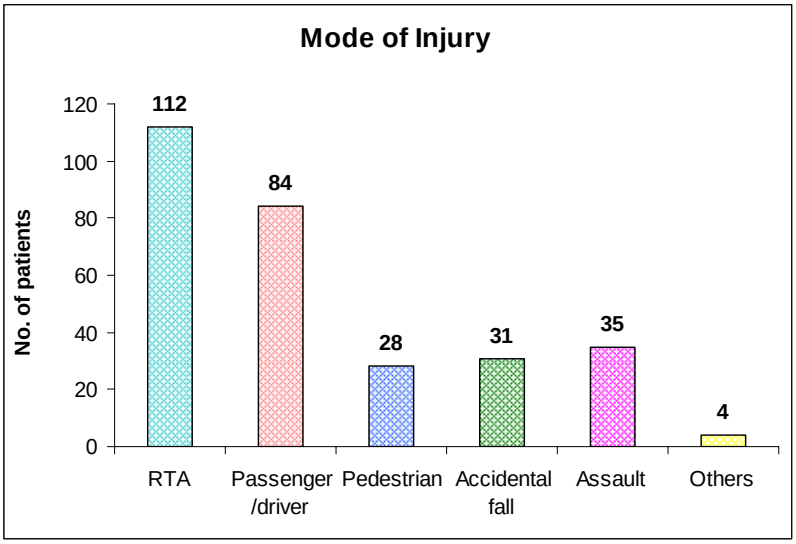
Gender	Number of patients	Percentage (%)
Male	112	61.54
Female	70	38.46



III. Mode of Injury

112 patients out of 182 gave a history of road traffic accident. Of them, 84 were passengers / drivers, 28 were pedestrians being hit by some vehicles. History of helmet wear was given only in 8 out of 84 patients. 31 Patients gave a history of accidental fall and 35 patients gave a history of assault. 4 gave history of injury at work place.

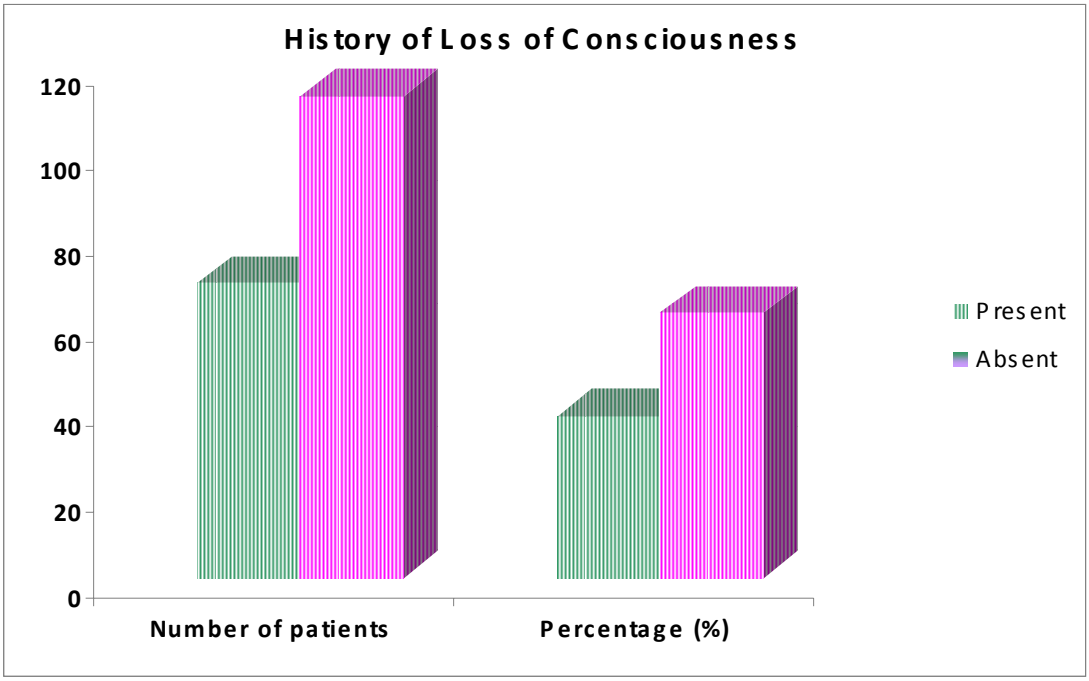
Mode of injury	Number of patients	Percentage (%)
RTA	112	61.54
Passenger /driver	84	75
Pedestrian	28	25
Accidental fall	31	17.03
Assault	35	19.23
Others	4	2.20



IV. History of loss of consciousness [LOC]

Out of 182 patients, only 69 patients gave a history of loss of consciousness.

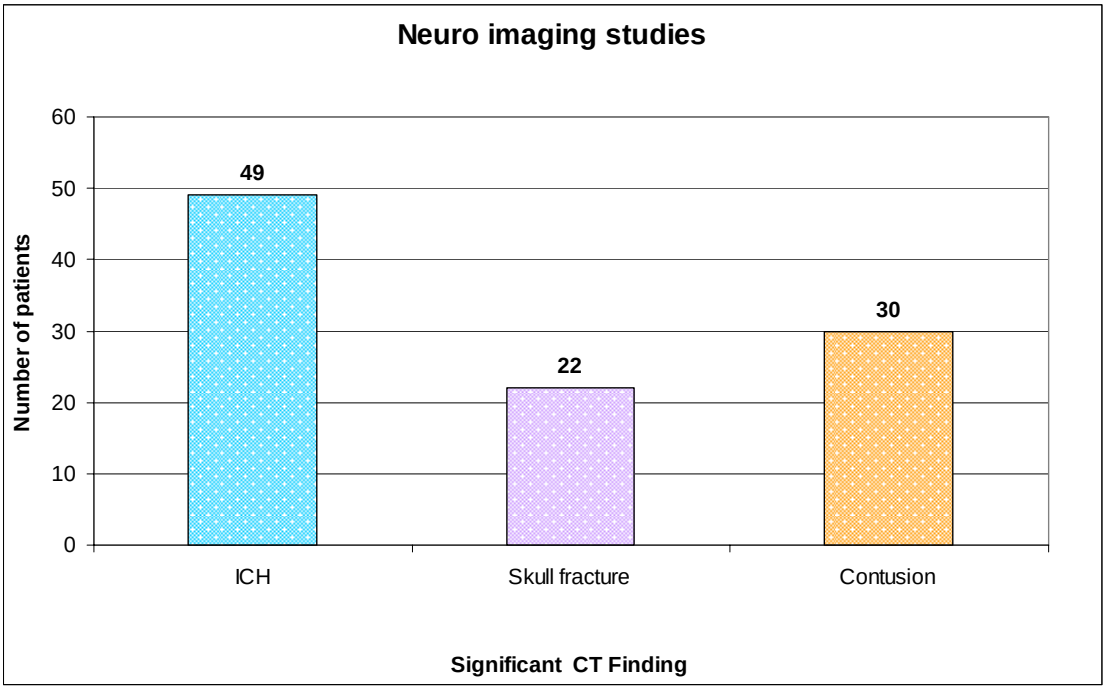
History of LOC	Number of patients	Percentage (%)
Present	69	37.94
Absent	113	62.09



IV. Neuroimaging studies

83 out of 182 patients had some finding in CT brain.

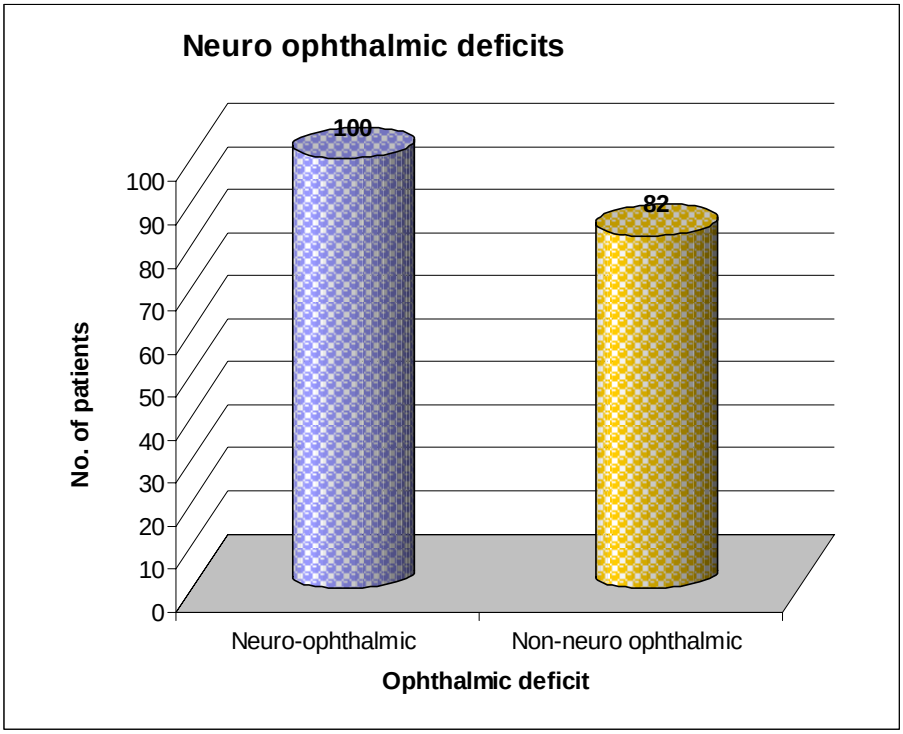
CT finding	Number of patients	Percentage (%)
Any significant present	**83**	**45.6%**
1. ICH	49	59.03
2. Skull fracture	22	26.50
3. Contusion	30	36.14



VI. Neuro ophthalmic deficits

Of 182 patients, only 100 patients had neuroophthalmic deficits on detailed examination. Other had non-neuro-opthalmic deficits like lid laceration, refractive error minor anterior segment injury. They were treated symptomatically and given reassurance.

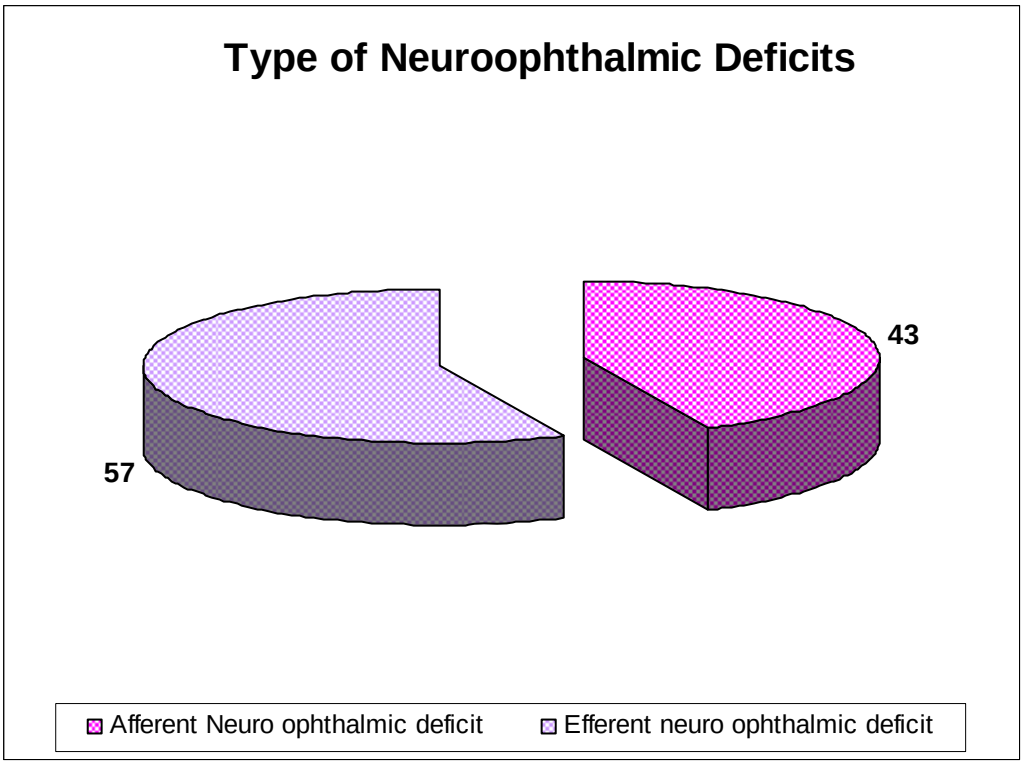
Ophthalmic deficit	Number of patients	Percentage (%)
Neuro-ophthalmic	100	54.94
Non-neuro ophthalmic	82	45.05



VII. Type of Neuro ophthalmic deficits

Out of 100 patients with neuro ophthalmic deficits, 43 patients had afferent deficits and 57 patients had efferent deficits.

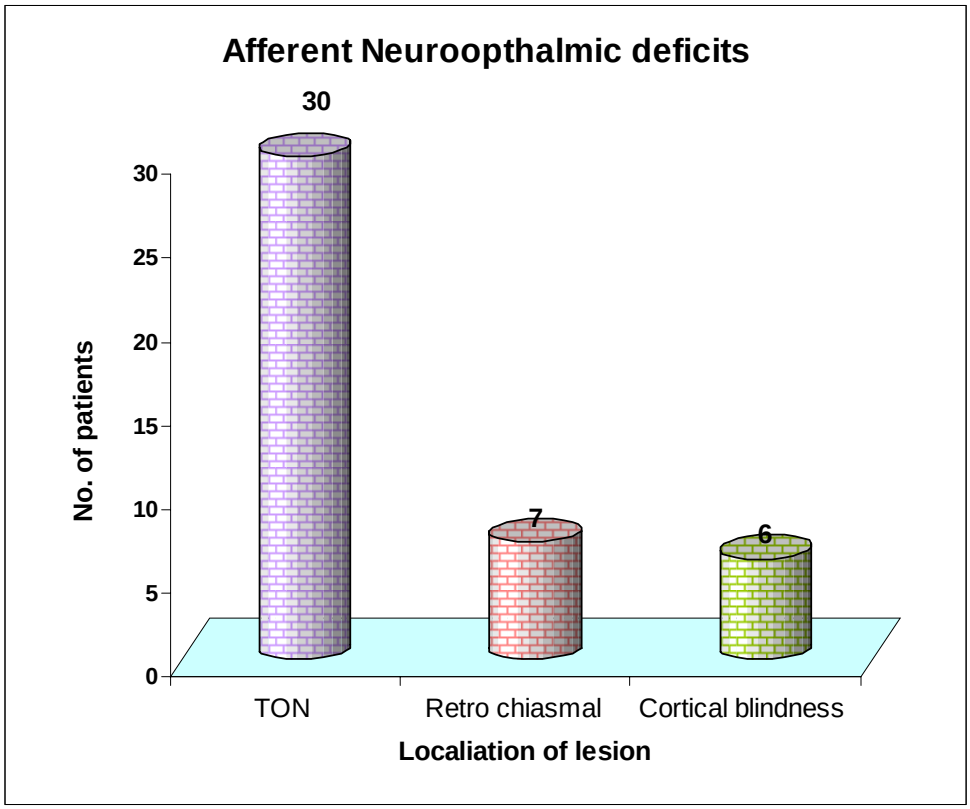
Afferent Neuro ophthalmic deficit	43
Efferent neuro ophthalmic deficit	57



VIII. Afferent neuroophthalmic deficits

30 out of 43 patients had traumatic optic neuropathy. Majority were of indirect type. They had unilateral visual defect with RAPD. 7 patients had findings suggestive of retro chiasmal lesion. 6 patients had cortical blindness.

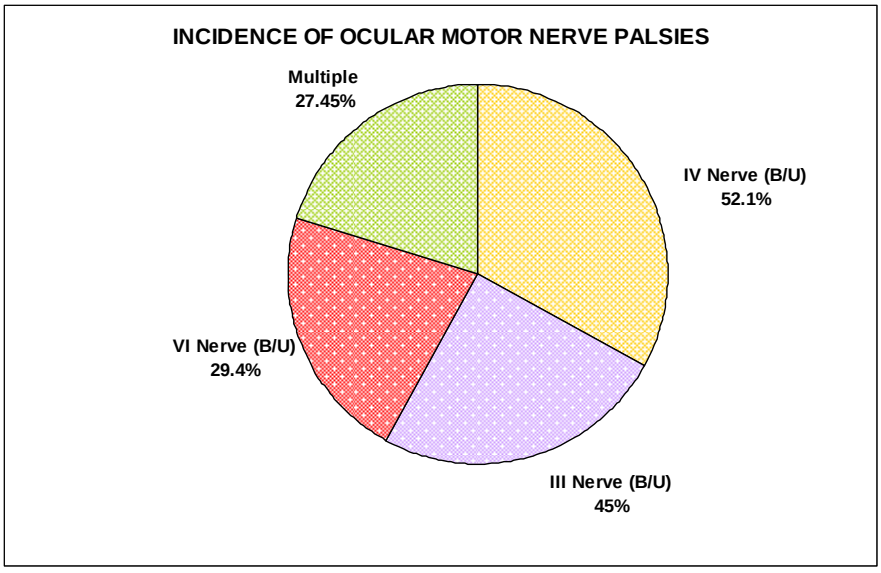
Location of lesion	Number of patients	Percentage (%)
TON	30	69.76
Retro chiasmal	7	16.28
Cortical blindness	6	13.95



IX. Ocular motor nerve palsies

51 patients out of 57 had some ocular motor nerve palsies. The most common cranial nerve injured was IV nerve followed by III nerve. VI nerve was least commonly affected. Maximum number of bilateral cases were seen in IV nerve injury. Multiple cranial nerve palsies were also seen whose incidence nearly equals VI cranial nerve palsy.

Ocular motor palsy	Number of patients	Percentage (%)
III Nerve (B/U)	23 (2/23)	45
IV Nerve (B/U)	27 (10/27)	52.1
VI Nerve (B/U)	15 (4/15)	29.40
Multiple	14	27.45



X. Other efferent neurological deficits

One patient had convergence insufficiency. Two patients had supranuclear gaze palsies and three patients had horner's syndrome.

Other efferent deficits	Number of patients	Percentage (%)
Convergence insufficiency	1	1.75
Horner's syndrome	3	5.20
Supranuclear gaze palsy	2	3.50

XI. Recovery pattern after 3 months of follow up

Recovery pattern was poor in patients with TON and retrochiasmal injury. Patients with cortical blindness usually showed good recovery.

Recovery pattern was very poor in multiple cranial nerve palsies.

Interestingly, 4 cases of III nerve injury showed features of aberrant degeneration.

Deficit	FR	PR	NR
TON	2	7	21
Retrochiasmal	-	5	2
Cortical blindness	4	1	1
III	6	10	7
IV	8	12	7
VI	7	5	3
Multiple	0	7	7
Convengence in sufficiency	-	1	-
Horners	2	-	1
Supra nuclear gaze palsy	-	-	2

RESULTS

1. Of the 182 cases of head injury, the age range was 2-70 years, with the maximum number of patients in the age group of 20-40 yrs constituting more than 60% of the total.
2. Males were more commonly affected than female with a M:F ratio of 1.6:1.
3. Road traffic accidents was the commonest mode of injury in our study. This constitutes 61% of the cases.
4. Only 38% of head injury patients gave a history of loss of consciousness.
5. Of the 182 patients, 83 patients had abnormal finding in CT brain. Majority of the patient with CT abnormality had intracranial hemorrhage (59%). They had III or IV nerve palsies. Basilar skull fracture was associated with bilateral VI nerve palsies.
6. Abnormal neuro ophthalmic findings were seen in 100 out of 182 patients. The rest of the patients had non-neuroophthalmic problems.
7. Efferent pathway deficit constituted 57% and more common than afferent pathway deficit (43%).
8. Traumatic optic neuropathy was the commonest afferent pathway deficit constituting 70%.

9. Of the ocular motor nerve palsies, IV nerve injury was the commonest injury (52.1%) followed by III nerve injury.
10. IV nerve palsy was quite often bilateral.
11. Only 29 patients showed full recovery 4 out of 6 patients with cortical blindness recovered fully. Aberrant regeneration was seen in 4 patients of III nerve injury in 3 months of follow up.

DISCUSSION

The incidence of motor vehicle accidents has dramatically increased in the past three decades. This is due to the mischievous behaviour of the adolescents and young adults in whom awareness of helmet wear while riding is lacking. Drunken driving is also one of the major causes of head injury in India.

MODE OF INJURY:

Motor vehicle accident was the commonest mode of injury in the present series constituting about 61%.Only 9.5% of cases gave history of helmet wear.This is comparable to various studies conducted in the past.

COMPARISION OF MOST COMMON

MODE OF INJURY

STUDIES	MOST COMMON MODE OF INJURY
Jacobi et al	45.6%
Keane	97.9%
Sabates et al	57%
Lepore	88.3%
Mariak et al	91.7%
Van Stavern et al	59.8%
Present series	61%

AGE GROUP :

Age range studied was 2 – 70 years. Maximum number of cases were in the age group of 20 – 40 years.

COMPARISION OF AGE GROUP STUDIED

STUDIES	AGE RANGE(Years)
Sabates et al	5 - 74
Lepore	8 - 66
Mariak et al	28 - 85
Keane	15 - 52
Van Stavern et al	2 - 86
Kowal	13 - 38
Present series	2 - 70

SEX DISTRIBUTION:

Men were more commonly affected than women.In the present series ,out of 182 patients,112 patients were males and 70 were females.

COMPARISION OF SEX DISTRIBUTION

STUDIES	SEX DISTRIBUTION
Jacobi et al	M = 471; F = 270
Sabates et al	M = 125; F = 56
Lepore	M = 28; F = 32
Mariak et al	M = 7; F = 5
Van Stavern et al	M = 203; F = 123
Present series	M = 112; F = 70

HISTORY OF LOSS OF CONSCIOUSNESS:

Loss of consciousness is not associated with any of the neuro ophthalmic deficits in the present series. A similar finding was seen by Van Stavern et al in his study.

COMPARISION OF HISTORY OF LOSS OF CONSCIOUSNESS

HISTORY OF LOC	Van Stavern et al	Present series
Present	39.9%	37.94%
Absent	60.1%	62.09%

MOST COMMON AFFERENT DEFICIT:

Traumatic optic neuropathy (TON) was the most common afferent deficit in the present series. Of them indirect optic nerve trauma constituted majority of cases (74.5%) . This is comparable with many of the studies. But it contradicts the study by Van Stavern et al which shows the retro chiasmal defects as the most common afferent deficit.

COMPARISION OF THE

MOST COMMON AFFERENT DEFICIT

STUDIES	MOST COMMON AFFERENT DEFICIT
Keane	**TON**
Kowal	**TON**
Moster et al	**TON**
Van Stavern et al	**Retro chiasmal defects**
Present series	**TON**

MOST COMMON EFFERENT DEFICIT:

Commonest cause of efferent pathway deficit was IV cranial nerve injury in the present series.

COMPARISION OF THE

MOST COMMON EFFERENT DEFICIT

STUDIES	MOST COMMON EFFERENT DEFICIT	INCIDENCE (%)
Jacobi et al	III Cr. N	5.1
Keane	III Cr. N	28.1
Sabates et al	IV Cr..N	40
Kowal	IV Cr..N	24
Lepore	IV Cr..N	33.3
Mariak et al	III Cr. N	50
Moster et al	III Cr. N	33
Van Stavern et al	IV Cr..N	51.2
Present series	IV Cr..N	52.1

INCIDENCE OF III CRANIAL NERVE PALSY:

The incidence of third cranial nerve palsy in the present series was 45%.Of them 8.69% of cases were bilateral.

COMPARISION OF THE

INCIDENCE OF III CRANIAL NERVE PALSY

STUDIES	INCIDENCE OF III CRANIAL NERVE PALSY(%)
Jacobi et al	15.9
Keane	28.1
Sabates et al	33
Kowal	10
Lepore	28.3
Mariak et al	50
Moster et al	33
Van Stavern et al	46.4
Present series	45

INCIDENCE OF IV CRANIAL NERVE PALSY:

The incidence of fourth cranial nerve palsy in the present series was 52.1%.About 37% of cases were bilateral.

COMPARISION OF THE

INCIDENCE OF IV CRANIAL NERVE PALSY

STUDIES	INCIDENCE OF IV CRANIAL NERVE PALSY(%)
Keane	**19.8**
Sabates et al	**40**
Kowal	**24**
Lepore	**33.3**
Mariak et al	**17**
Moster et al	**28**
Van Stavern et al	**51.2**
Present series	**52.1**

INCIDENCE OF VI CRANIAL NERVE PALSY:

The incidence of sixth cranial nerve palsy in the present series was 29.40%. Bilateral sixth nerve palsy was seen in 26.67%.

COMPARISION OF THE INCIDENCE OF VI CRANIAL NERVE PALSY

STUDIES	INCIDENCE OF VI CRANIAL NERVE PALSY(%)
Keane	26
Sabates et al	27
Kowal	10
Lepore	11.7
Mariak et al	17
Moster et al	20
Van Stavern et al	25
Present series	26.67

INCIDENCE OF MULTIPLE CRANIAL NERVE PALSY:

Multiple cranial nerve palsy was seen in 27.45% patients.This is comparable to study by Van Stavern et al.

COMPARISION OF THE

INCIDENCE OF MULTIPLE CRANIAL NERVE PALSY

STUDIES	INCIDENCE OF MULTIPLE CRANIAL NERVEPALSY(%)
Van Stavern et al	22.6
Present series	27.45

SIGNIFICANT NEURO IMAGING ABNORMALITY:

In the present series , only 45.6% of patients had evidence of injury in CT. Van Stavern et al found significant abnormality in 47.3% of head injury patients. There was a correlation between intra cranial haemorrhage and third and fourth cranial nerve palsies. Basillar skull fracture was associated with sixth cranial nerve palsies. Asimilar inference was given by Van Stavern et al in his study.

COMPARISION OF SIGNIFICANT

NEURO IMAGING ABNORMALITY

NEURO IMAGING ABNORMALITY	Van Stavern et al	Present series
Absent	52.7%	54.4%
Present	47.3%	45.6%
ICH	62.1%	59.03%
Skull fracture	24.2%	26.50%
Contusion	28.7%	36.14%

CONCLUSION

The following conclusions are made from the above observations:

1. Head injury is more common in the age group of 20-40 years.
2. Males are more commonly affected than females.
3. The most common mode of head injury is road traffic accident.
4. Loss of consciousness is not associated with any neuroophthalmic deficits.
5. Efferent visual pathway deficit is more common than afferent visual pathway deficit.
6. Commonest efferent visual pathway deficit is IV nerve injury.
7. Commonest afferent visual pathway deficit is indirect traumatic optic neuropathy.
8. Bilateral injury is more common with IV nerve injury.
9. Recovery pattern of visual pathway deficits are not very good inspite of timely management.
10. The presence of significant neuroimaging abnormality, particularly intracranial haemorrage is significantly associated with III and IV nerve injuries.

11. Even in the absence of any neuroimaging abnormality, the prevalence of neuro-ophthalmic deficits is high.
12. Meticulous awareness of traffic rules, helmet wear, giving up of drunken drive are the safety measures to avoid road traffic accidents and to prevent head injury for, “prevention is always better than cure”.

PROFORMA

Neuro ophthalmic manifestations of head injuries

Date:

1. Name
2. Age/sex
3. Occupation & address
4. I.P. No.
   Head injury No. Unit: N.S. I/II/III
   Eye OP NO.
   Neuro-opthal clinic No.
5. Date of Admission: Discharge:
6. Ocular Complaints/duration:
7. Information related to injury:
   1. Mode of injury:
      RTA: pedestrian/non motorized/two wheeler / closed vehicle
      Fall: Home / workplace <6ft/6ft
      Domestic accident
      Industrial /blast/falling object
   2. Assault : Blunt/sharp weapon
   3. Under alcohol

      Associated injuries: (specify)
      Chest/abdomen / faced ENT / Spine / orthopaedic

8. Post-traumatic : LOC duration
   Amnesia / vomiting / giddiness
   Fits: type & number
   ENT bleed/rhinorrhoea / otorrhoea
   Vision / diplopia in immediate period.
   (if symptomatic at examination)

9. Glasgow coma scale:
   On admission: On exam: trend:
10. Vital Signs : P/T/R/B.P
11. History of Ocular problems: Onset/ Course / duration
    1)
    2)
    3)
12. Detailed description of salient complaint:

    EXAMINATION : Right Eye Left Eye

13. Proptosis / enophthalmos
14. Orbital margins
15. Lids / periocular area
16. Conjunctiva / cornea
17. AC/ins
18. Pupil size / shape
    - Reflex
      Direct Consensual
    - RAPD Grade
    - Near synkinesis
19. Lens
20. Sensation
    - Corneal
    - Lids and face

OPTIC NERVE AND VISUAL PATHWAY

21. Vision (Bedside)
    [FC and near card]
22. Refr. Error and BCVA
23. Colour perception

- Simple red target
- Ishihara's

24. Brightness sense

    (Compare eyes)

25. Fields

    - Confrontation
    - Tangent screen / perimeter (target size)

26. **EXTRA OCULAR MOVEMENTS**

    Tropia:

    - ET/XT/HT
    - 1° deviation
    - Sec. Deviation

27. Nystagmus

28. Diplopia charting

29. Conjugate movements
    (Suranuclear)

30. Oculocephalic / Oculorestibular reflex
    [only if gaze affected]

31. Oculokinetic nystagmus

32. Fundus examination

    Media
    Disc margins
    Size / Shape / colour
    Swelling
    Peripapillary & background
    Vessels
    Macula

33. Slit lamp examination

34. Hertel's exophthalmometry

35. Tension

36. General examination

37. CNS: Higher functions
Cranial nerves
Motor system
Sensory system
DTR and plantar reflex
Gait and coordination

INVESTIGATIONS

38. Skull x-ray

39. CT scan Brain
Skull bone # location / type
(1) Haemorrhage EDH / SDH/SAH/ICh
(2) Parenchyma
- Oedema
- Contusion

(3) Compression
- Ventricule
- Midline shift

(4) Pneumocephalus

Review CT Brain :

40. CT Scan orbit
Orbital wall #:
Optic nerve:
Intra canalicular
Intra orbital
Soft tissue; haematoma / emphysema
Globe:

41. Provisional Diagnosis:

1. Ophthalmic / Neuro ophthalmic
2. Neurosurgical

42. Coincident surgical problems

Chest
Abdomen

Spine

Orthopaedic

43. Medical Problems : Diabetes / hypertension / uremia
44. Progress & Result :
45. Follow up : in Ophthalmology OPD

MASTER CHART															
S.No.	**Name**	**Age**	**Sex**	**IP No.**	**Mode of Injury**	**H/o LOC**	**Pre Symptoms**	**Laterality**	**VA**	**Ant Segm**	**Pupil**	**Fundus**	**Clinical Diagnosis**	**Neuro imaging**	**Follow up**
1	Raju	30	M	012467	RTA	-	Eye pain diplopia	LE	6/6	NAD	RTL	NAD	6th N palsy	SDH	FR
2	Karpagam	27	F	011354	RTA	-	Z	BE	6/9	NAD, Rt.Homo. hemianopia	RTL	NAD	Retrochias mal injury	NAD	PR
3	Ponni	29	F	019765	RTA	+	Diplopia	BE	6/6	NAD Diplopia	RTL	NAD IV	IV N Palsy	Basilar #	NR
4	Palsamy	60	M	018564	Fall	+	Def .Vn	RE	HM	SCH	RAPD	NAD II	II N Palsy	NAD	PR
5	Natraj	32	M	013856	Assault	-	Def .Vn	RE	No PL	SCH	RAPD	NAD II	II N Palsy	ICH (Rt) FL	NR
6	Hemesh	16	M	017621	RTA	-	Pain, HA	RE	6/6	NAD Diplopia	NAD	NAD	IV N Palsy	NAD	PR
7	Ramesh	30	M	017629	RTA	+	Def .Vn	LE	6/36	NAD	RAPD	NAD	II N Palsy	NAD	NR
8	Eswaran	53	M	018719	Fall	+	Diplopia	LE	6/24	NAD	DIL. NRTL	NAD	III N Palsy	Contusion MB	FR
9	Manoharan	35	M	017257	RTA	+	Diplopia	BE	6/6	SCH	DIL. NRTL	NAD	III N Palsy	Hge MB	PR
10	Muthukumar	36	M	016999	Assault	-	Def .Vn	LE	6/60	NAD	RAPD	Papilledema	II N Palsy	NAD	FR
11	George	17	M	019274	Assault	-	Eye pain	(BE)	6/9	NAD	RTL	NAD	IV N Palsy	Basilar #	PR
12	Palpandi	27	M	023595	RTA	-	Ptosis, HA	RE	6/6	SCH	DIL. NRTL	NAD	III, IV N Palsy	Hge MB	PF
13	Suresh	8	M	025496	Fall	-	Def .Vn	(BE)	6/60	NAD	RTL	NAD	Cortic. Blindness	NAD	FR
14	Paramasivam	47	M	025325	RTA	+	Diplopia	LE	6/24	NAD, Diplopia	DIL. NRTL	Papilledema	III, IV, VI, palsy	Hge MB	NR
15	Pandi	29	M	024431	RTA	+	Def .Vn	RE	PL	SCH	RAPD	Disc edema	II N Palsy	NAD	NR
16	Kannan	19	M	024371	RTA	-	Ptosis, HA	RE	6/12	NAD	DIL. NRTL	NAD	III, IV N Palsy	Hge MB	NR
17	Abdul	30	M	025146	RTA	-	Def .Vn	BE	6/6	SCH	RTL	NAD	Supra.Nu. gaze palsy	Contusion (Rt) FL	NR
18	Gurusamy	54	M	026714	Work place	+	Diplopia	RE	6/24	NAD, Diplopia	RTL	NAD	IV N Palsy	NAD	FR
19	Geetha	37	F	027738	RTA	-	Diplopia	RE	6/6	NAD, Diplopia	RTL	Papilledema	VI N Palsy	Basilar #	PR
20	Madhivanan	48	M	023998	Fall	+	Def .Vn	(BE)	3/60	NAD	RTL	NAD	Cortic. Blindness	Contusion occi. Lobe	PR
21	Anbunathan	47	M	026620	Assault	-	Def .Vn	LE	No PL	SCH	RAPD	NAD	II N Palsy	# O.Canal	NR
22	Chellammal	32	F	029995	RTA	+	Def .Vn	BE	6/12	NAD, (Lt) Homo Hemianopia	RTL	NAD	Retrochias mal lesion	Contusion Rt. PL	PR

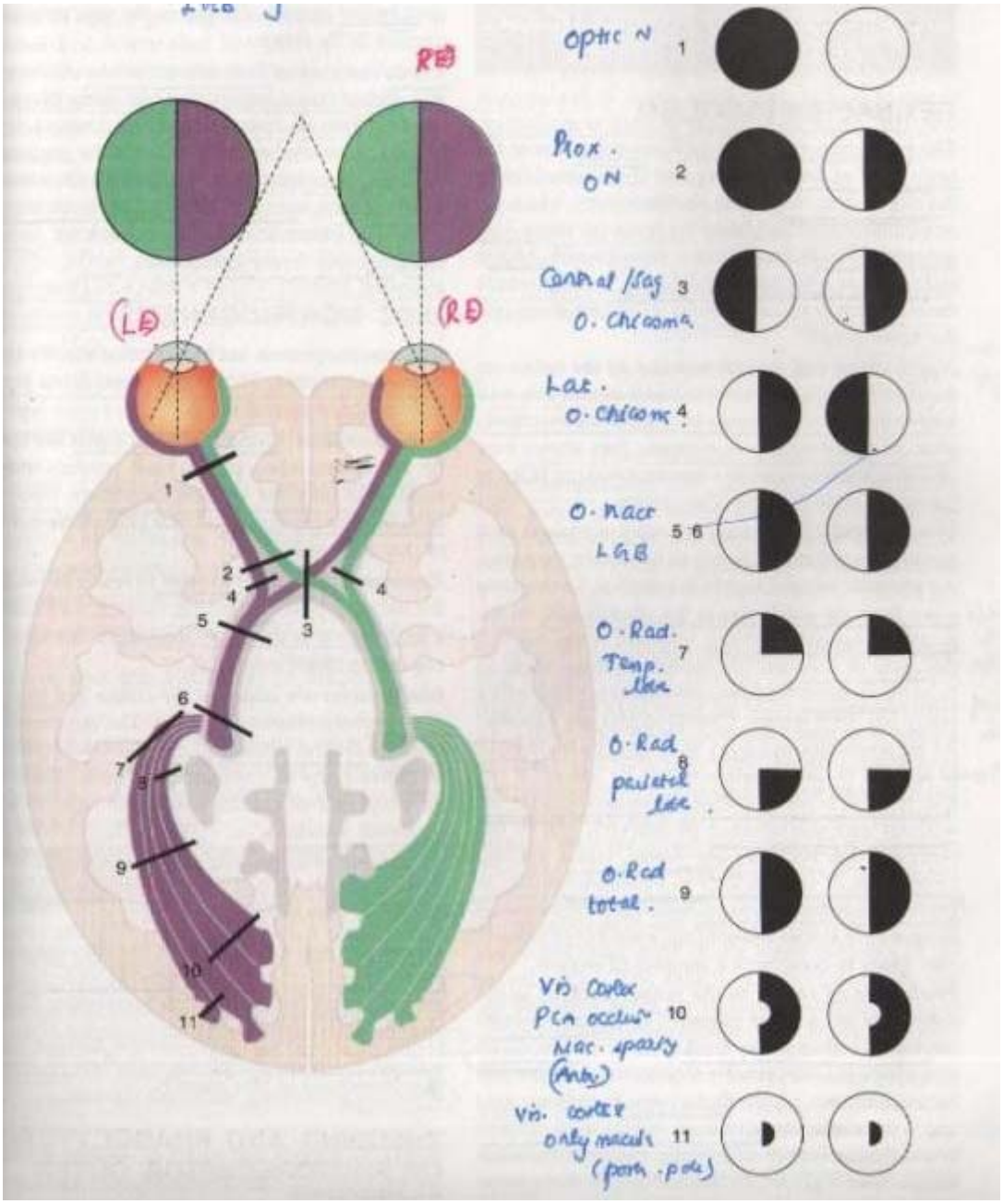
S.No.	Name	Age	Sex	IP No.	Mode of Injury	H/o LOC	Pre Symptoms	Laterality	VA	Ant Segm	Pupil	Fundus	Clinical Diagnosis	Neuro imaging	Follow up
23	Mary	67	F	029998	RTA	-	Diplopia	BE	6/24	NAD	RTL	NAD	IV N Palsy	Basilar #	PR
24	Mari Raj	34	M	028718	Fall	-	Ptosis	(LE)	6/6	NAD, Diplopia	DIL. NRTL	NAD	III N Palsy	NAD	PR
25	Gokul	18	M	030019	RTA	+	Def .Vn	RE	2/60	Ecchymosis, Proptosis	RAPD	Disc edema	II N Palsy	RBH	FR
26	Rajesh	28	M	031286	RTA	-	Diplopia	RE	6/6	NAD	RTL	NAD	IV N Palsy	NAD	NR
27	Surya	26	F	031481	RTA	+	Diplopia	RE	6/6	SCH	RTL	NAD	VI N Palsy	NAD	FR
28	Pitchai	57	M	032167	Fall	-	Pain	RE	6/12	Ptosis	DIL. NRTL	NAD	IV, III N Palsy	SDH	NR
29	Amsavalli	28	F	032817	RTA	-	Ptosis	LE	6/6	SCH, Ptosis	DIL. NRTL	NAD	III N Palsy	NAD	PR
30	Angayarkanni	31	F	032264	RTA	+	Def .Vn	RE	6/60	NAD	RAPD	Disc edema	II N Palsy	Ons. Haematoma	PR
31	Govindan	26	M	038192	RTA	-	Def .Vn	LE	HM	NAD	RAPD	NAD	II N Palsy	NAD	PR
32	Mookayee	38	F	039937	Assault	-	Diplopia	RE	6/6	NAD	RTL	NAD	IV N Palsy	NAD	PR
33	Rohini	27	F	038252	Assault	+	Ptosis	LE	6/6	SCH, Ptosis	DIL. NRTL	NAD	III N Palsy	EDH	PR
34	Sundaram	30	M	039733	RTA	-	Diplopia	(RE)	6/9	NAD	RTL	NAD	IV N Palsy	NAD	PR
35	Kanthammal	46	F	039898	Fall	-	Def .Vn	RE	6/60	SCH	RAPD	NAD	II N Palsy	Ons. Haematoma	PR
36	Saira banu	32	F	039921	Assault	+	Def .Vn	LE	No PL	NAD	RAPD	NAD	II N Palsy	# O.Canal	NR
37	Hari haran	32	M	045395	RTA	+	ptosis	LE	6/12	SCH, Ptosis	DIL. NRTL	NAD	III N Palsy	Basilar #	PR
38	Anjali	26	F	046954	RTA	-	Headache Def.vn	LE	6/60	NAD	RAPD	NAD	II N Palsy	EDH	NR
39	Roseline	30	F	046817	RTA	-	Diplopia	BE	6/6	SCH, Diplopia	RTL	NAD	IV N Palsy	Basilar #	PR
40	Kanthimathi	52	F	041110	Fall	-	ptosis	RE	6/24	NAD	DIL. NRTL	NAD	III N Palsy	NAD	FR
41	Thangam	29	M	047421	Assault	+	Def .Vn	BE	6/18	NAD, (Rt) homo hemianopia	RTL	NAD	Retrochias mal injury	ICH (RT) PL	PR
42	Mariappan	44	M	045753	Assault	-	Def .Vn	RE	6/60	SCH	RAPD	NAD	II N Palsy	Contusion (RT) FL	NR
43	Abirami	28	F	045579	RTA	-	HA, eye pain	RE	6/6	SCH, enophthal	Miotic SRTL	Papilledema	Horner's	ICH MB	FR

S.No.	Name	Age	Sex	IP No.	Mode of Injury	H/o LOC	Pre Symptoms	Laterality	VA	Ant Segm	Pupil	Fundus	Clinical Diagnosis	Neuro imaging	Follow up
44	Alagar	28	M	048596	RTA	+	Diplopia	RE	6/6	NAD, Diplopia	RTL	NAD	IV N Palsy	ICH MB	NR
45	Velammal	62	F	051001	RTA	-	Def.vn, HA	(RE)	No PL	NAD	RAPD	NAD	II N Palsy	SDH	NR
46	Suresh	26	M	051214	RTA	-	Ptosis, eye pain	(LE)	6/6	SCH, Ptosis	DIL. NRTL	NAD	III IV N Palsy	ICH MB	NR
47	Amsuvalli	32	F	051875	Fall	+	Ptosis	RE	6/9	SCH	DIL. NRTL	Papilledema	III, IV N Palsy	SDH	PR
48	Balaji	15	M	051815	RTA	-	Def .Vn	BE	6/60	NAD	RTL	NAD	Cortical blindness	Contusion OL	FR
49	Mani	34	M	053976	RTA	-	HA, Ptosis	RE	6/9	Ptosis, Diplopia	DIL. NRTL	NAD	III, IV, N palsy	SDH	NR
50	Amaravathy	35	F	058807	Fall	+	HA	LE	6/9	Ptosis, Enophthal	Miotic SRTL	NAD	Horneis	NAD	PR
51	Karuppayee	50	F	059555	Assault	-	Def .Vn	LE	6/60	NAD	RAPD	NAD	II N Palsy	# O.Canal	NR
52	Saravanan	26	M	059785	RTA	+	HA, Diplopia	LE	6/12	NAD	RTL	NAD	IV N Palsy	EDH	PR
53	Gomathy	45	F	061110	Assault	+	Def .Vn	RE	No PL	SCH	RAPD	Retin.he	II N Palsy	SAH	NR
54	Vijay	15	M	061094	RTA	-	HA	BE	6/6	NAD Diplopia	RTL	NAD	IV N Palsy	EDH	FR
55	Raju	29	M	062137	Fall	-	Diplopia	BE	6/6	NAD, Diplopia	RTL	NAD	VI N Palsy	NAD	FR
56	Manikandan	29	M	063936	RTA	+	Def .Vn	(RE)	6/60	NAD	RAPD	NAD	II N Palsy	EDH	NR
57	Chellam	28	F	063381	RTA	+	Headache	(BE)	6/6	NAD, Diplopia	RTL	NAD	IV N Palsy	Basilar #	PR
58	Abdul	9	M	063993	RTA	-	Def .Vn	BE	3/60	NAD	RTL	NAD	Cortical Blindness	NAD	FR
59	Gururaj	31	M	068235	RTA	-	Diplopia	LE	6/6	NAD, Diplopia	RTL	NAD	IV N Palsy	Hge MB	NR
60	Thangam	22	F	069595	RTA	+	Def .Vn	LE	6/36	NAD	RAPD	NAD	II N Palsy	NAD	NR
61	Rajapandi	63	M	069641	RTA	-	Def .Vn	LE	6/60	SCH	RAPD	NAD	II N Palsy	NAD	NR
62	Arunkumar	24	M	067728	RTA	-	Headache, Diplopia	RE	6/12	NAD, Diplopia	RTL	NAD	IV N Palsy	NAD	PR
63	Siddique	49	M	068975	Work place	+	Ptosis, HA	RE	6/12	Ptosis, Diplopia	DIL. NRTL	NAD	III N Palsy	NAD	FR
64	Kanaga	24	F	069143	RTA	-	Def. Vn (LE) 1/2 of VF	LE	6/12	NAD, (Lt) homo.hemianopia	RTL	NAD	Retrochias mal injury	Contusion Rt. PL	NR
65	Banu	17	F	067149	RTA	+	HA, Ptosis	RE	6/6	SCH, Diplopia	DIL. NRTL	NAD	III, IV N Palsy	Basilar #	NR
66	Eswaran	25	M	068789	RTA	-	Headache	LE	6/6	NAD, Diplopia	RTL	NAD	VI N Palsy	NAD	NR

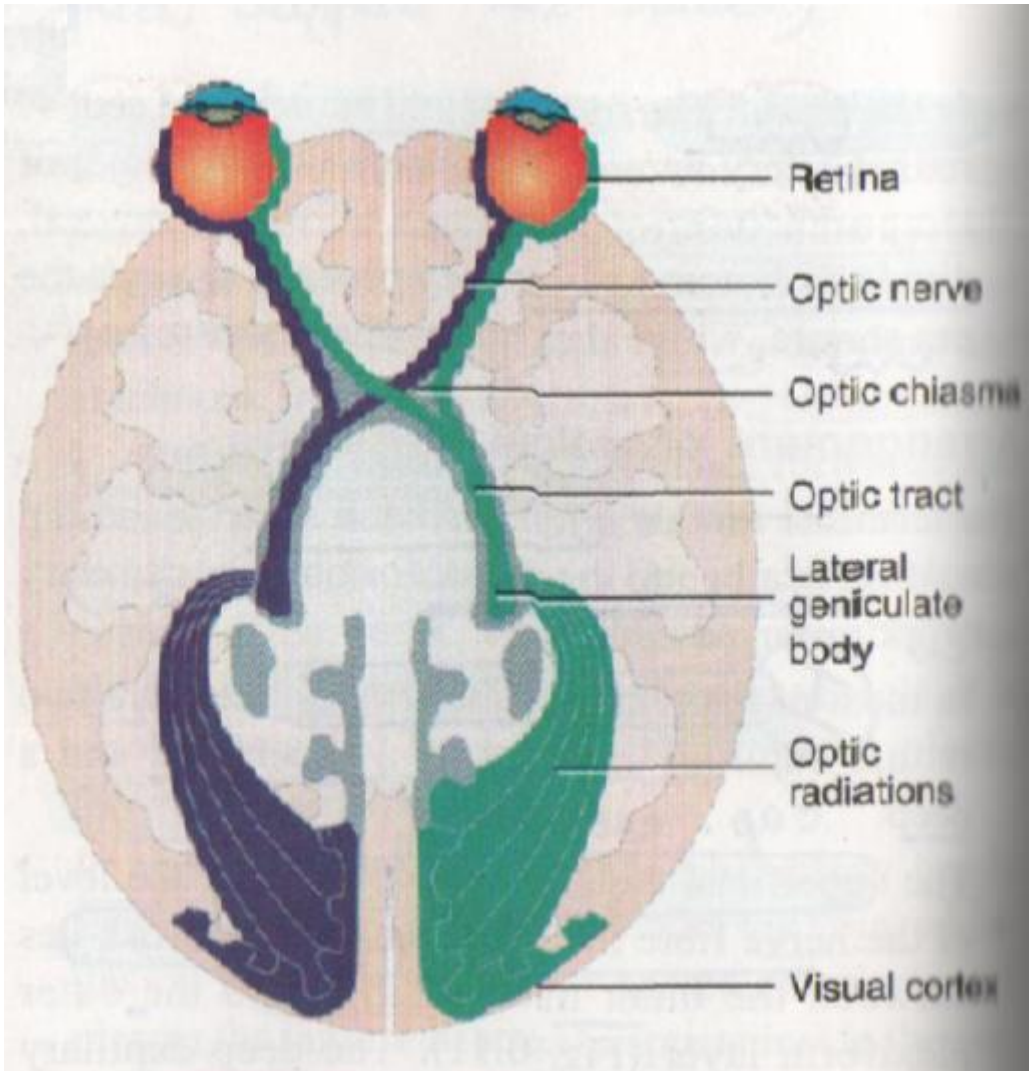
S.No.	Name	Age	Sex	IP No.	Mode of Injury	H/o LOC	Pre Symptoms	Laterality	VA	Ant Segm	Pupil	Fundus	Clinical Diagnosis	Neuro imaging	Follow up
67	Ammaponnu	32	F	062134	RTA	+	Diplopia	BE	6/6	SCH, Diplopia	RTL	NAD	IV N Palsy	Basilar #	NR
68	Anbu	37	F	059891	Assault	-	Def .Vn	RE	6/60	NAD	RAPD	NAD	II N Palsy	NAD	NR
69	Saravanan	30	M	058735	RTA	-	HA	RE	6/6	Homo.hemianopia, with macular sparing	RTL	NAD	Retrochiasmal injury	ICH (RT) OL	NR
70	Periakaruppan	56	M	069494	Fall	+	Def .Vn	BE	6/36	NAD	RTL	NAD	Cortic. Blindness	Contusion OL	FR
71	Palaniammal	45	F	068172	Assault	-	Def .Vn	(LE)	3/60	NAD	RAPD	NAD	II N Palsy	NAD	NR
72	Paul	25	M	068986	RTA	+	Def .Vn	RE	No PL	Ecchymosis, SCH	RAPD	NAD	II N Palsy	Contusion (Rt) FL	PR
73	Pitchai	25	M	068198	RTA	-	Ptosis	BE	6/9	NAD, Diplopia	DIL. NRTL	NAD	III N Palsy	Hge MB	FR
74	Priya	15	F	068279	RTA	-	Def .Vn	LE	6/60	NAD	RAPD	NAD	II N Palsy	NAD	NR
75	Shankar	49	M	071012	Fall	-	Diplopia	LE	6/6	NAD, Diplopia	RTL	NAD	VI N Palsy	NAD	FR
76	Selvamahesh	33	M	071921	Fall	+	HA, Diplopia	RE	6/6	SCH, Diplopia	RTL	Papilledema	VI N Palsy	Basilar #	PR
77	Sharada	54	F	072026	RTA	+	Def .Vn	LE	No PL	NAD	RAPD	NAD	II N Palsy	NAD	NR
78	Madhusudhan	36	M	072148	Assault	-	Ptosis	RE	6/9	SCH, Diplopia, Ptosis	DI NRTL	NAD	III, IV N Palsy	Contusion MB	PR
79	Karthik	33	M	073763	Assault	-	Headache	RE	6/12	Enophth, Ptosis	Miotic SRTL	NAD	Horner's	Basilar #	NR
80	Natarajan	27	M	074040	RTA	-	Diplopia	RE	6/6	NAD, Diplopia	RTL	NAD	IV N Palsy	NAD	FR
81	Pooja	9	F	074168	RTA	+	Def .Vn	BE	1/60	NAD	RTL	NAD	Cortic. Blindness	OL Hge	NR
82	Kokila	29	F	074327	RTA	-	Def .Vn	RE	6/6	NAD, Diplopia	RTL	NAD	VI N Palsy	Contusion Rt. TL	FR
83	Sundar	16	M	074565	Fall	-	HA, Diplopia	BE	6/9	SCH, Diplopia	RTL	Papilledema	VI N Palsy	Basilar #	PR
84	Ramesh	16	M	078807	RTA	-	Ptosis , HA	RE	6/6	NAD	DIL. NRTL	NAD	III, IV N Palsy	Hge MB	PR
85	Ayyavoo	67	M	071015	RTA	+	Def .Vn	RE	PL	NAD	RAPD	NAD	II N Palsy	# O.Canal	PR
86	Pothom Ponnu	28	F	073381	Assault	-	Def .Vn	RE	6/60	NAD	RAPD	NAD	II N Palsy	NAD	FR
87	Ranjani	28	F	076286	RTA	-	Def.Vn, HA	RE	3/60	SCH	RAPD	NAD	II N Palsy	SAH	NR
88	Kumarasamy	42	M	079413	Assault	-	HA, Ptosis	LE	6/6	SCH	DIL. NRTL	NAD	III N Palsy	Basilar #	PR

S.No.	Name	Age	Sex	IP No.	Mode of Injury	H/o LOC	Pre Symptoms	Laterality	VA	Ant Segm	Pupil	Fundus	Clinical Diagnosis	Neuro imaging	Follow up
89	Muthu	26	M	079942	RTA	-	Def .Vn	BE	6/12	NAD, Rt Homo hemianopia	RTL	NAD	Retrochiasmal injury	Contusion (Lt) PL	PR
90	Kuppusamy	55	M	079987	Fall	+	HA, Def. Vn	BE	6/18	NAD	RTL	Papilledema	Conv. Insufficiency	SDH	PR
91	Mariammal	25	F	079093	RTA	-	Diplopia	RE	6/6	NAD, Diplopia	RTL	NAD	IV N Palsy	Basilar #	PR
92	Renuka	25	F	078194	RTA	-	Def .Vn	LE	No PL	NAD	RAPD	NAD	II N Palsy	EDH	NR
93	Rajan	23	M	079211	RTA	+	Diplopia	RE	6/6	NAD, Diplopia	RTL	NAD	IV N Palsy	Contusion MB	FR
94	Babu	29	M	080463	RTA	-	Eye Pain, Ptosis	RE	6/12	SCH, Ptosis, Diplopia	DIL. NRTL	NAD	III N Palsy	Basilar #	PR
95	Velu	17	M	080781	RTA	-	HA, Diplopia	LE	6/6	NAD	RTL	NAD	IV N Palsy	EDH	FR
96	Mohamed Ali	69	M	080948	Fall	-	Headache	(BE)	6/36	SCH	miotic RTL	NAD	Supra.Nugaze palsy	Hge Pons	NR
97	Ganesan	32	M	082888	RTA	-	Def .Vn	(BE)	6/12	NAD (Lt) Homohemianopia	RTL	NAD	Retrochiasmal injury	ICH (RT) TL	NR
98	Murugavel	47	M	083892	Assault	+	Def .Vn	(RE)	1/60	NAD	RAPD	NAD	II N Palsy	NAD	PR
99	Alagar	30	M	084144	RTA	+	Ptosis	(LE)	6/12	NAD, Ptosis, Diplopia	DIL. NRTL	NAD	III, IV N Palsy	ICH MB	NR
100	Lakshmanan	35	M	085163	Assault	-	Def .Vn	RE	HM	NAD	RAPD	NAD	II N Palsy	NAD	NR

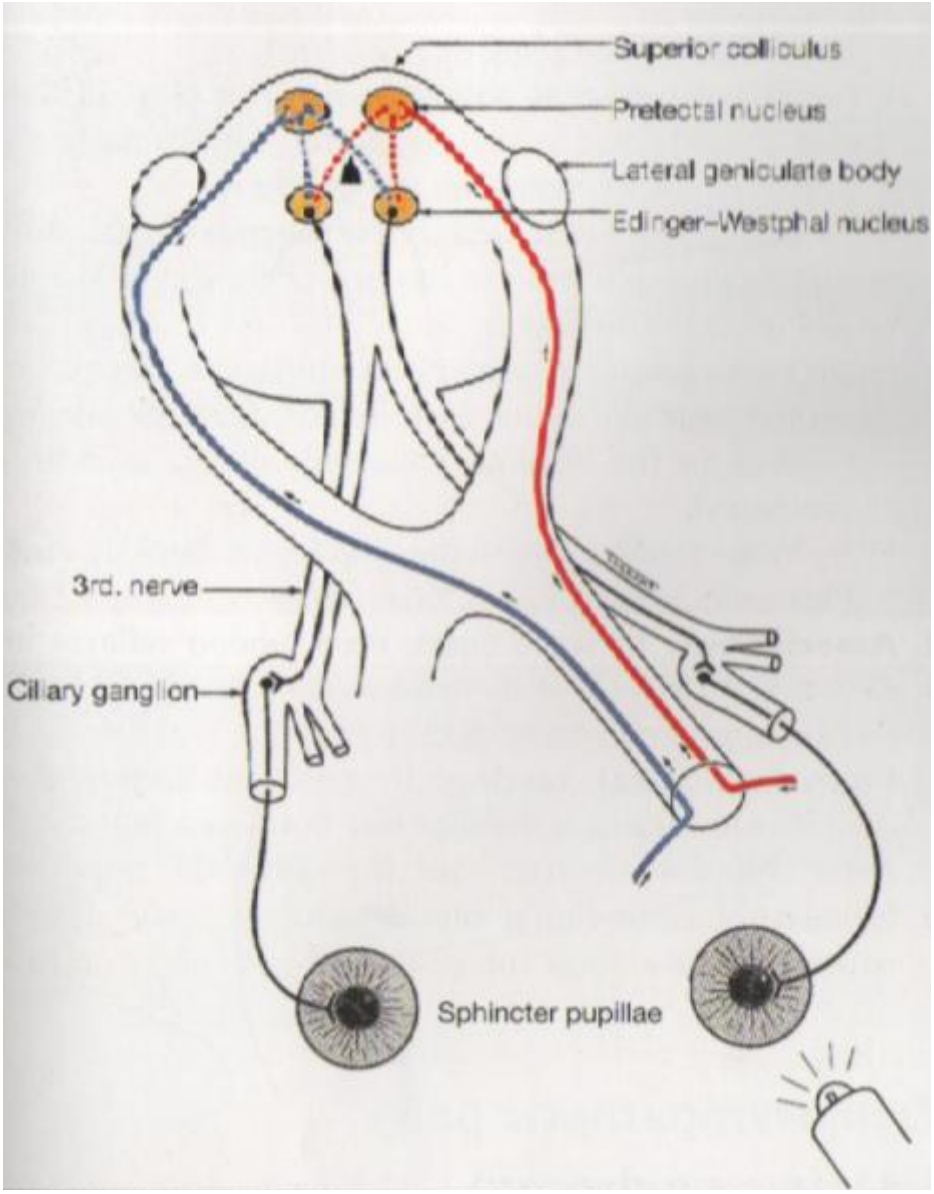
LESIONS AT VARIOUS LEVELS OF VISUAL PATHWAY AND CORRESPONDING VISUAL FIELD DEFECTS

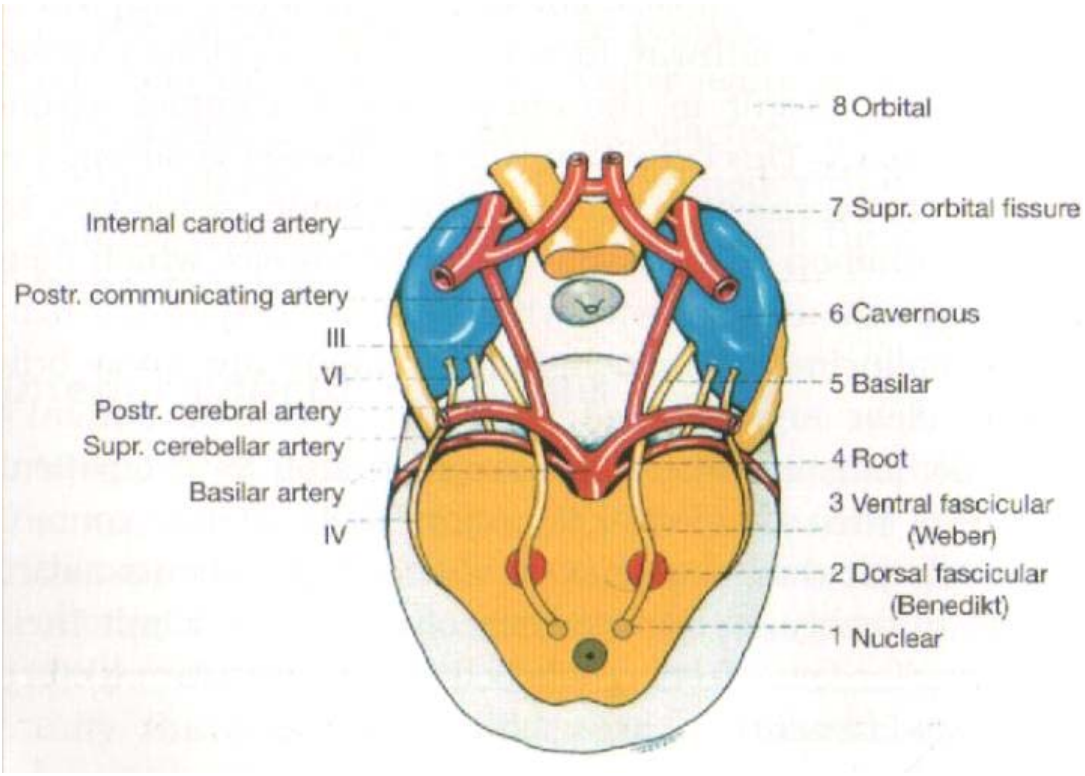


VISUAL PATHWAY

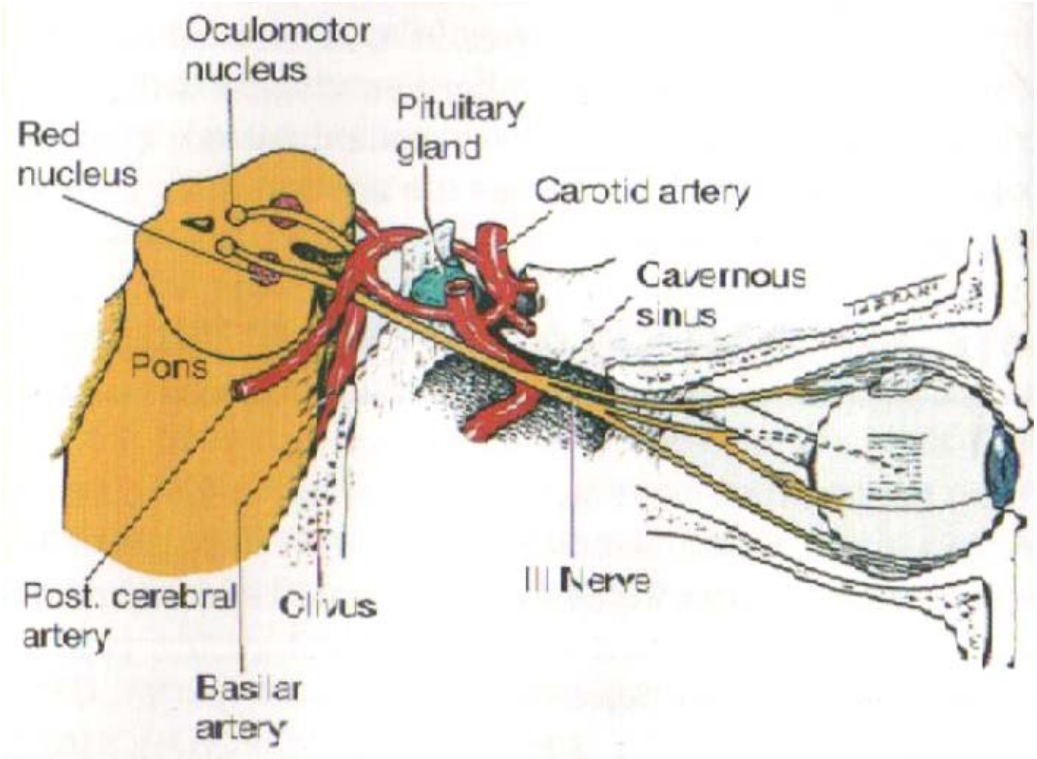


THE PATHWAY OF PUPILLARY LIGHT REFLEX

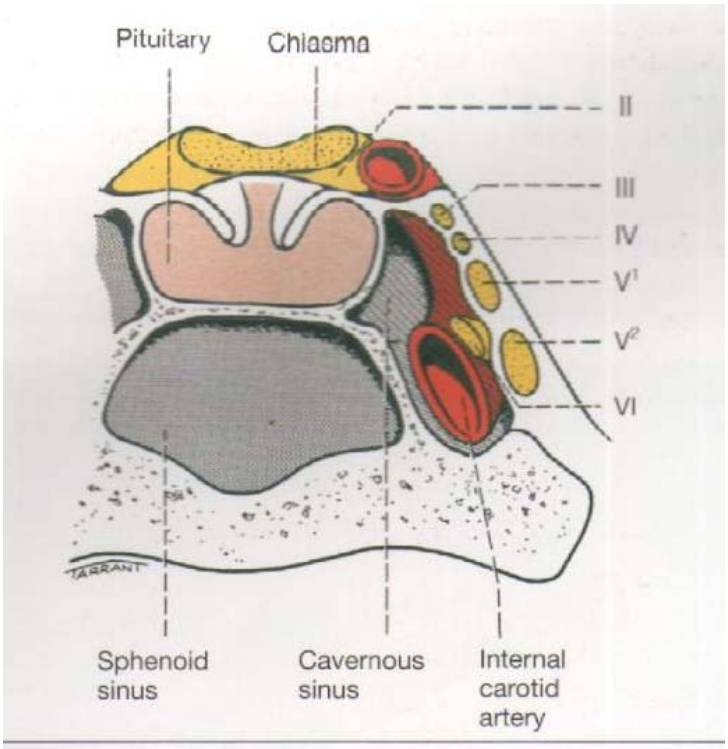




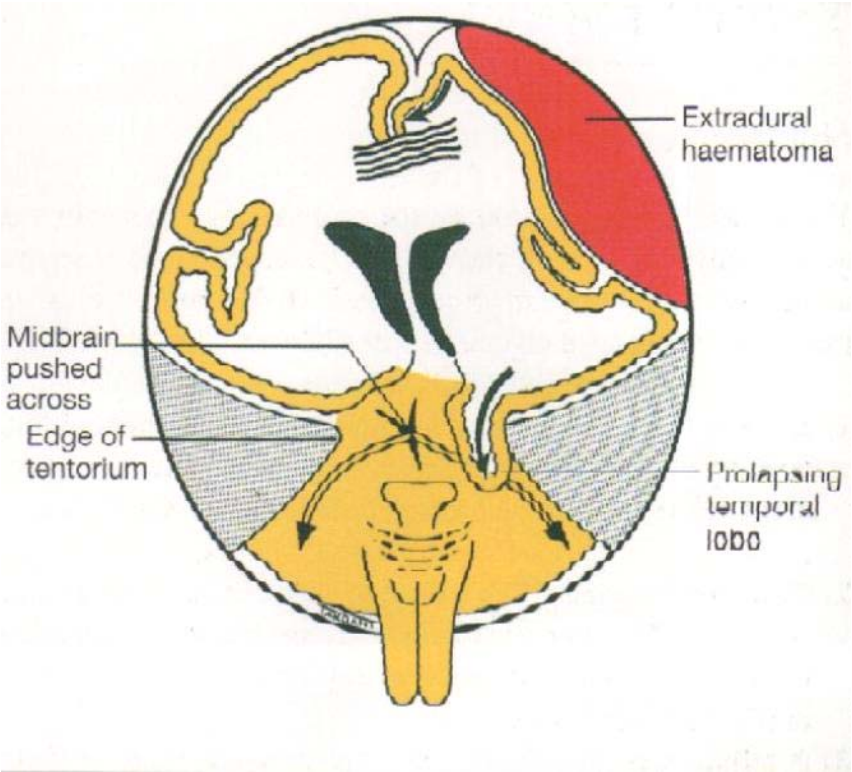
DORSAL VIEW OF THE COURSE OF THE THIRD NERVE



LATERAL VIEW OF THE COURSE OF THE THIRD NERVE

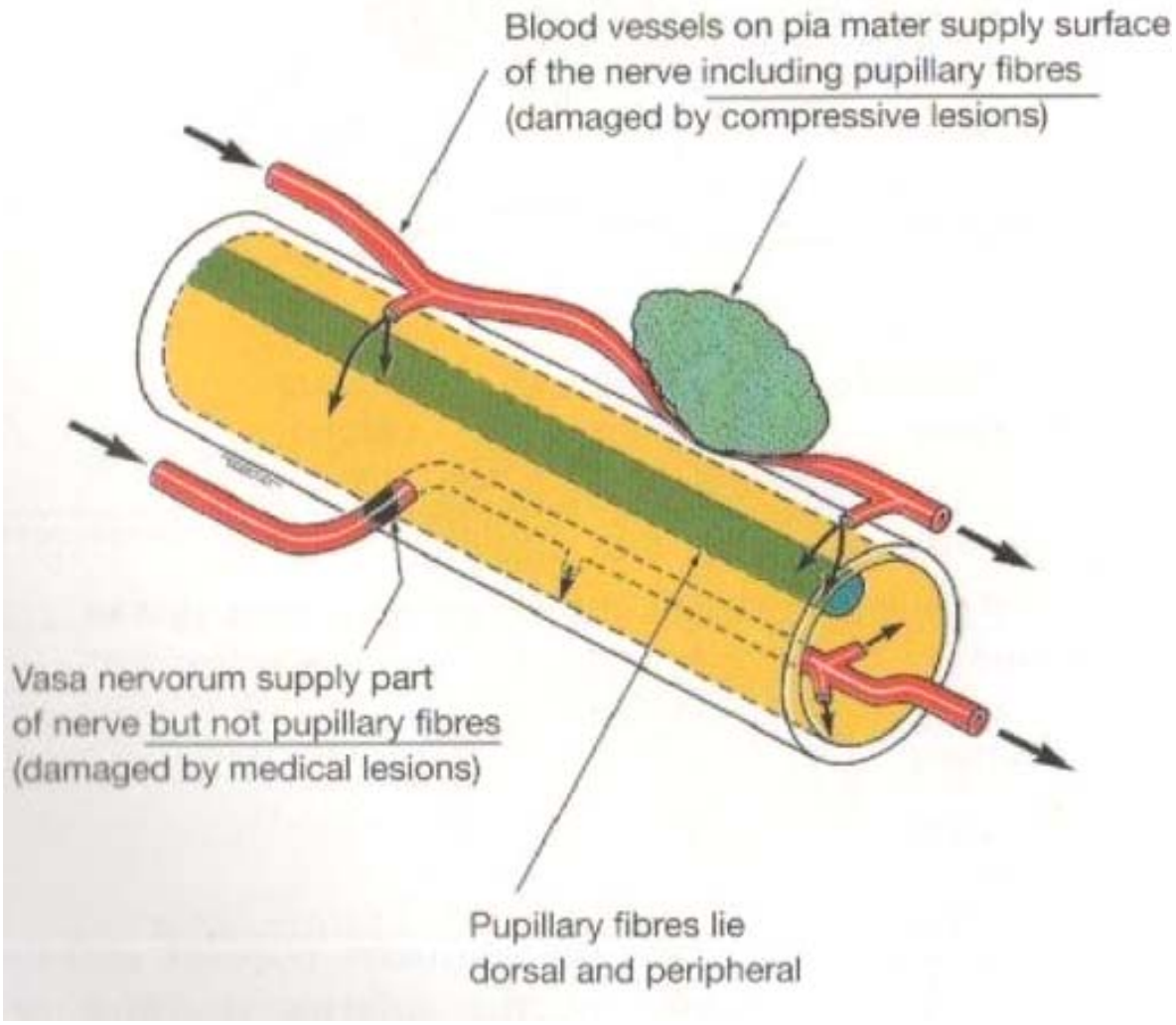


LOCATION OF THE CRANIAL NERVES IN THE CAVERNOUS SINUS VIEWED FROM BEHIND

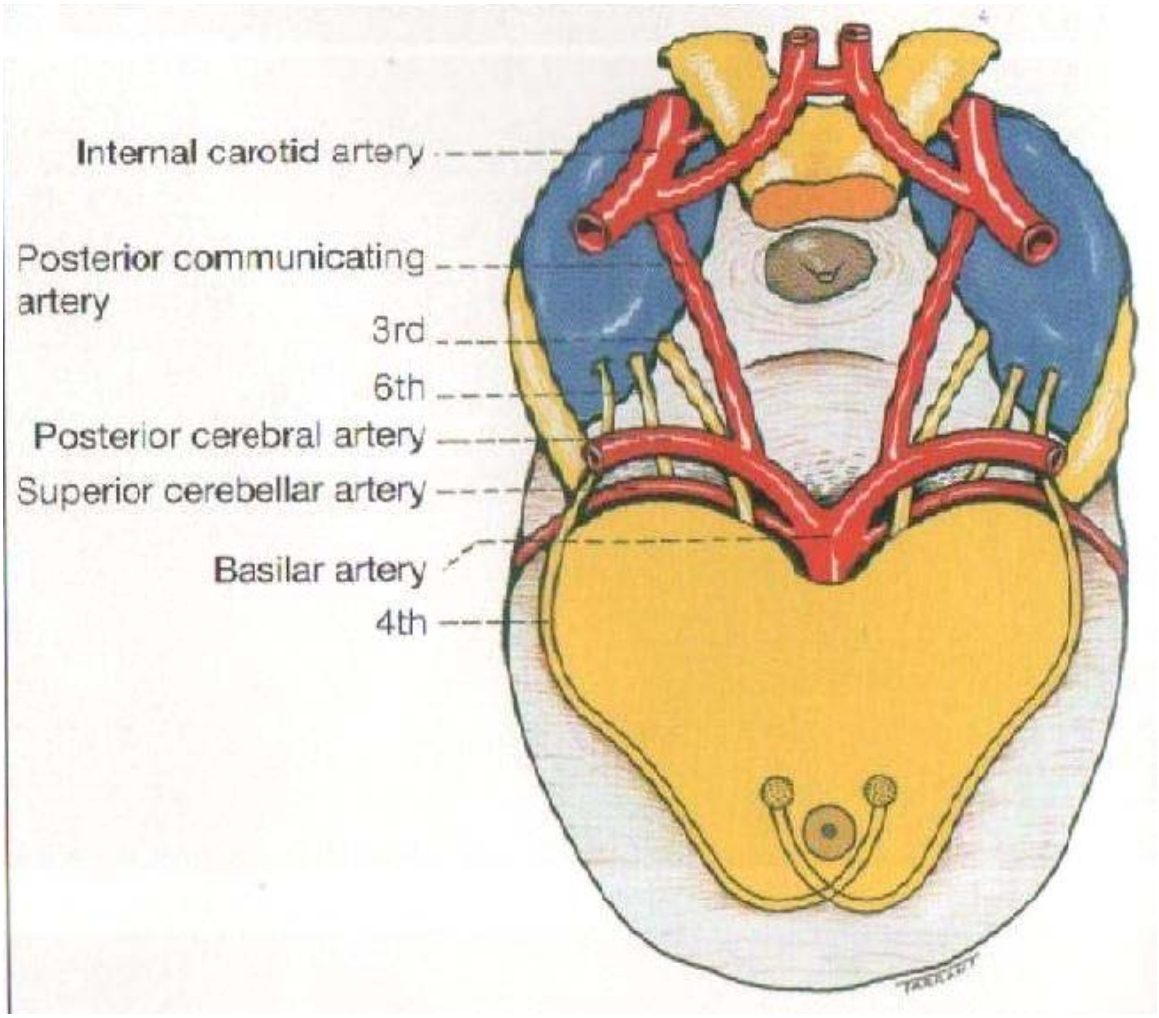


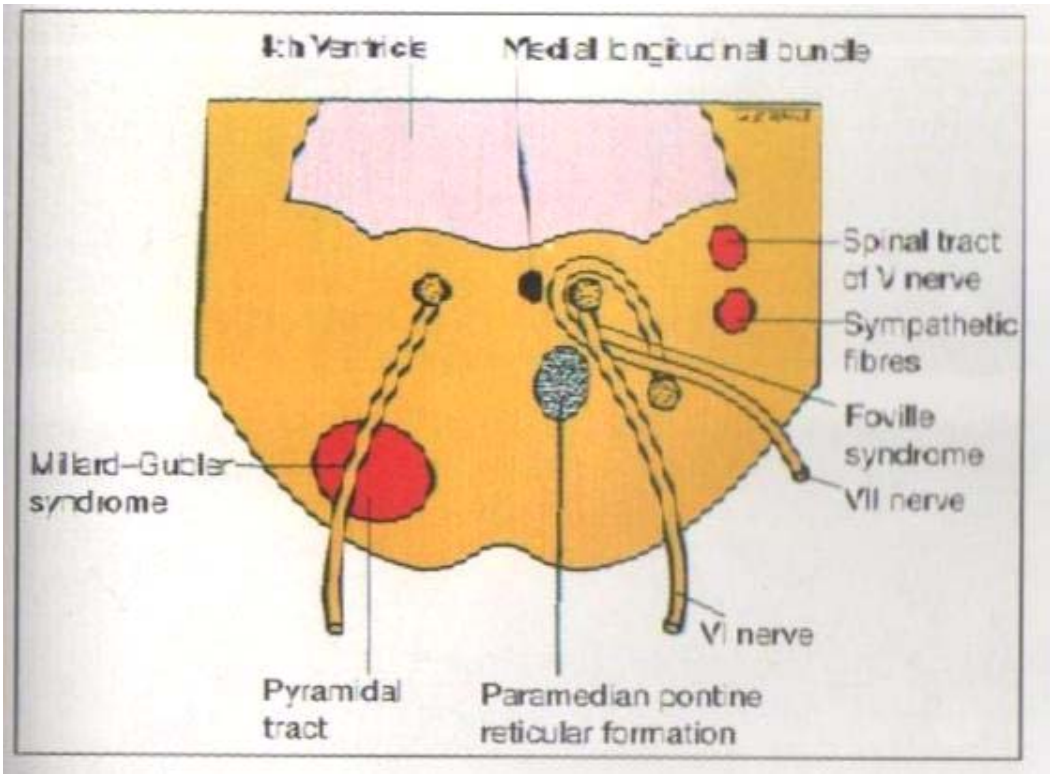
MECHANISM OF THIRD NERVE PALSY BY EXTRA DURAL HAEMATOMA

LOCATION OF PUPILLOMOTOR FIBRES WITHIN THE TRUNK OF THE THIRD NERVE

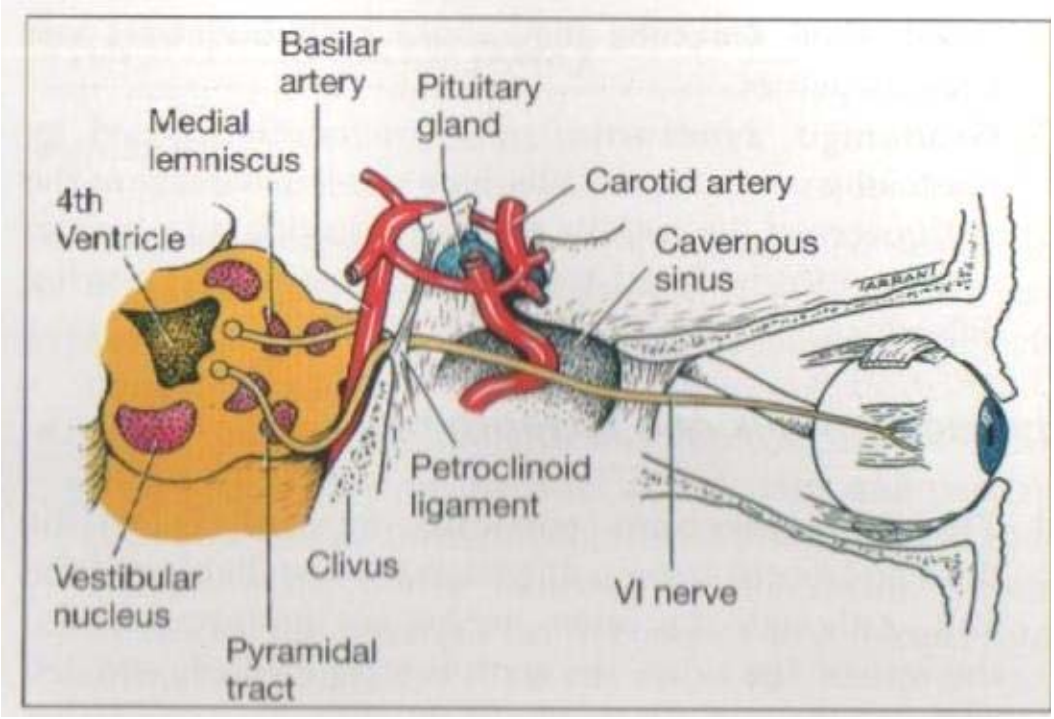


DORSAL VIEW OF THE COURSE OF THE FOURTH NERVE



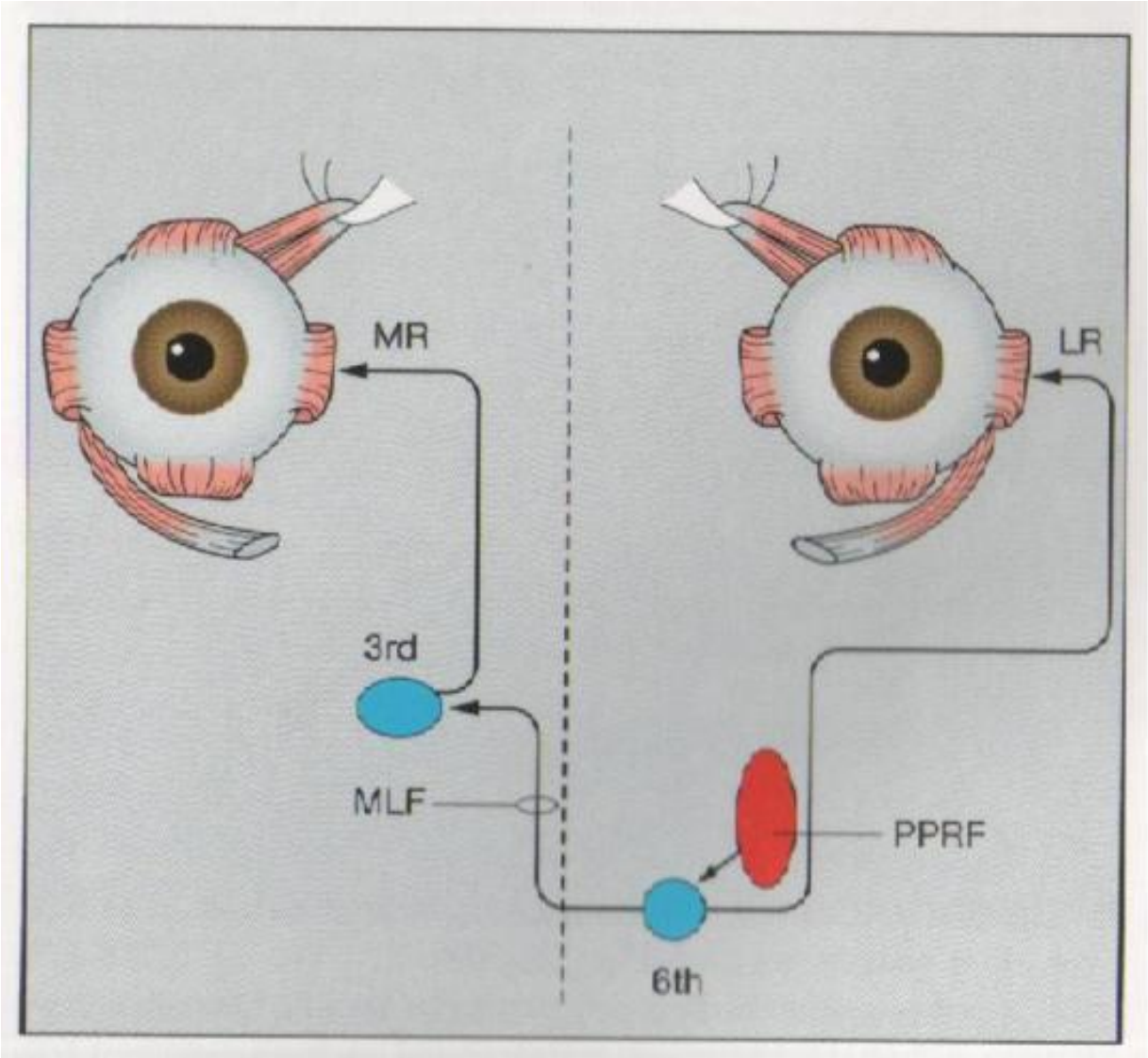


THE SIXTH NERVE NUCLEUS AT THE LEVEL OF THE PONS



LATERAL VIEW OF THE COURSE OF THE SIXTH NERVE

ANATOMICAL PATHWAYS FOR HORIZONTAL EYE MOVEMENTS

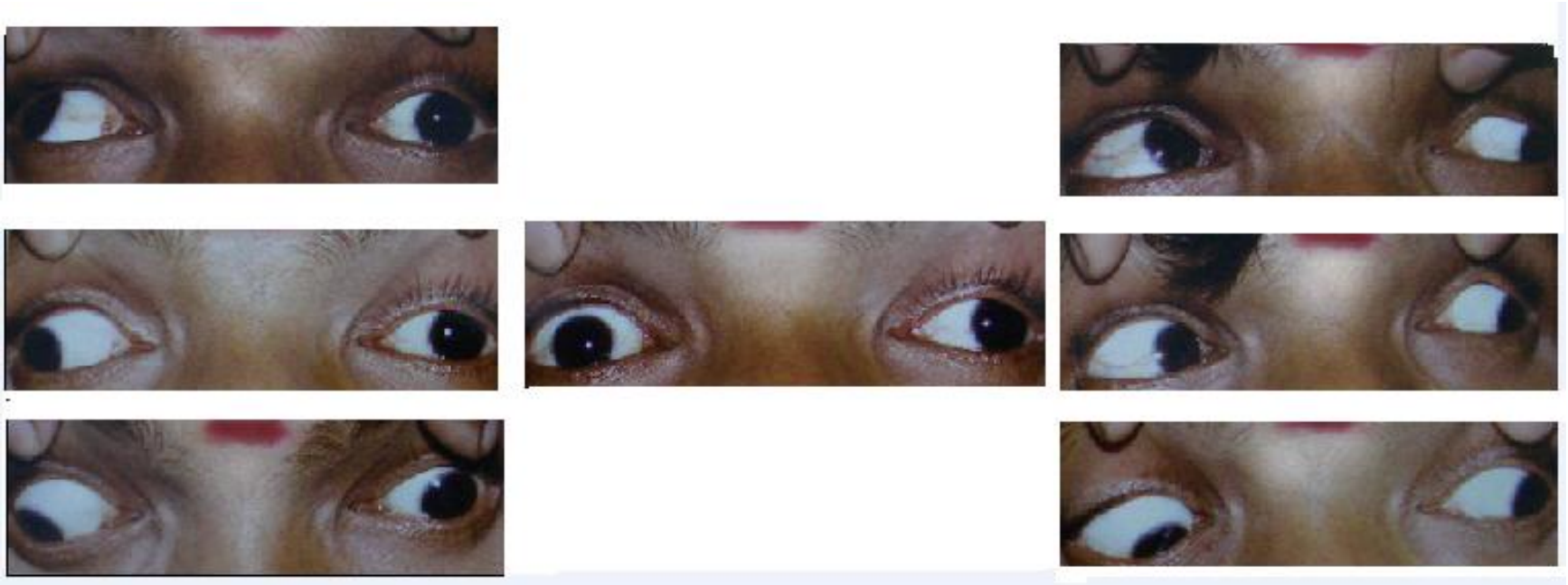


PPRF : Pontine Paramedian Reticular Formation
MLF : Medial Longitudinal Fasciculus
MR : Medial Rectus
LR : Lateral Rectus

OCULOMOTOR NERVE PALSY – LEFT EYE

BILATERAL ABDUCENS NERVE PALSY

MULTIPLE CRANIAL NERVE PALSY – LEFT EYE



TROCHLEAR NERVE PALSY – LEFT EYE